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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

Thirty-eighth Annual Meeting

THE AMERICAN ACADEMY OF PERIODONTOLOGY

November 4, 5, 6, 1954

Miami Beach, Florida

Spring Seminar

May 2, 3, 4, 1955

Ann Arbor, Michigan

FRANCIS J. FABRIZIO, D.D.S.

Chairman, Section on Periodontics, American Dental Association

Dr. Francis J. Fabrizio, as Chairman of the Section on Periodontics of the American Dental Association, has prepared an essay program to be given Monday, November 8 at 2 p.m. On his program will be Dr. B. S. Chaikin, Boston; Dr. Richard Chace, Orlando, Florida; Dr. J. Lewis Blass, New York; and Dr. Dorothea F. Radusch, Minneapolis.

Dr. Fabrizio is Associate Professor of Periodontology at the School of Dentistry, Georgetown University, and Civilian Consultant in Periodontia to the United States Naval Dental Corps, National Institute of Health and the United States Army.

His practice is limited to periodontics.

THE JOURNAL OF PERIODONTOLOGY

OCTOBER, NINETEEN HUNDRED FIFTY-FOUR

Modification of the Effect of Cortisone Upon Alveolar Bone by the Systemic Administration of Estrogen

by IRVING GLICKMAN, B.S., D.M.D.* AND GERALD SHKLAR, B.SC., D.D.S., M.S.,**
Boston, Mass.

IN a previous study[1] it was reported that, in white mice, repeated injections with cortisone (Cortone-Acetate, Merck) resulted in osteoporosis of alveolar bone and other bones which was characterized by a reduction in the number of osteoblasts and amount of osteoid matrix. The bone loss was attributed to a disturbance in the corticosteroid hormone balance responsible for the anabolic-catabolic equilibrium essential for the maintenance of bone. The above finding led to conjecture regarding the possibility of preventing or compensating for the osteoporotic effect of an excess of cortisone by the systemic administration of a substance which favors rather than inhibits bone formative activity. The availability of estrogenic hormone with a demonstrated capacity of stimulating osteogenic activity[2-7] enabled us to explore the aforementioned possibility.

EXPERIMENTAL PROCEDURE

Sixty young adult white mice (thirty males and thirty females) approximately 6 weeks old and weighing 18-24 gm. were divided into three groups of twenty animals each (10 males and 10 females) as follows: Group I (Cortisone group) received daily subcutaneous injections of 0.25 mg. cortisone acetate (Cortone acetate, Merck); Group II (Cortisone-Estrogen group) received daily subcutaneous injections of 0.25 mg. cortisone, and in addition received one weekly subcutaneous injection of 600 rat units of estradiol benzoate (Progynon B, Schering); Group III (Control group). Ten animals received daily subcutaneous injections of physiological saline, and ten were not injected.

The animals in all the groups were sacrificed at the end of a 35-day experimental period. The mandible, vertebrae, femur and all internal organs were then removed, fixed in 10 per cent formalin and prepared for microscopic study.

FINDINGS

Gross observations: Fourteen of the forty animals in groups I and II died in

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We wish to thank Schering Corporation for supplying us with the Progynon B, and Merck and Company for supplying us with the Cortone Acetate used in these experiments.

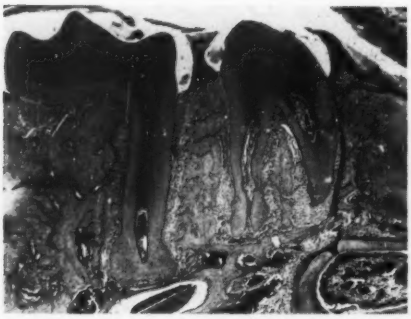

Fig. 1. Control animal. Mesio-distal survey section through the mandible showing the first and second molars and alveolar bone. (H.E. x 56)

the course of the experiment. At the end of the five week experimental period, the animals in the cortisone group were smaller than the controls and presented a roughening of the fur. The animals in the cortisone-estrogen group were somewhat smaller than the controls but larger than the cortisone injected animals.

MICROSCOPIC OBSERVATIONS

Alveolar Bone—Interdental Septum

Control animals: The distal surface of the bone between the first and second molars was smooth in outline and lined with a beadlike layer of osteoblasts (Fig. 1 and 2). In some areas, osteoblasts were partially embedded in newly formed osteoid. The mesial surface of the septum was irregularly indented and presented occasional multinucleated osteoclasts in lacunar indentations. At the crest of the septum, along the distal aspect there was newly formed osteoid with prominent embedded collagen fibers.

Cortisone Injected Animals: There was no evidence of any appreciable bone formation in relation to either the mesial or distal surface or the crest of the interdental septum (Fig. 3 and 4). The margins of both the mesial and distal surfaces presented an irregularly indented deeply staining border devoid of osteoblasts or osteoid. A saucer-like depression formed the crest.

Fig. 2. Control animal. Detailed section of Fig. 1 showing the interdental septum and periodontal membrane. Note the smooth distal surface (right) with adjacent osteoblasts, new bone formation, and imbedded collagen fibers at the crest. The mesial surface (left) is irregularly indented. Note the difference in the morphology of the fibroblasts of the periodontal membrane at the crest of the interdental septum compared with those in the remainder of the section. (H. E. x 290)

The height of the interdental bone was reduced. Within the bone of the septum, the osteocyte lacunae were larger and irregularly arranged when compared with the control. The appositional lines were more prominent and somewhat less regularly arranged than the control. There was an occasional area where endosteal bone forma-

Fig. 3. Cortisone injected animal. Mesio-distal survey section through the mandible, showing the first and second molars and alveolar bone. (H.E x 56)

tion was observed in relation to a medullary cavity, but for the most part, the medullary spaces were outlined by irregularly indented deeply staining margins.

Cortisone-Estrogen Animals: The interdental bone between the first and second molars differed from that of the cortisone injected animals in that it presented new bone formation at the crest (Figs. 5 and 6). This appeared as a section of osteoid and newly formed bone containing large spherical osteocytes. A multi-layered band of fibroblasts outlined the periphery of the newly formed bone. Osteoblasts were seen immediately adjacent to the bone and in some areas were partially embedded in osteoid. The mesial and distal surfaces of the remainder of the septum were for the most part irregularly indented and outlined by a deeply staining irregular border. In isolated areas a bead-like layer of osteoblasts was also observed along the distal surface in relation to pale staining osteoid. Vessel channels extending from the periodontal membrane into the septum were lined by prominent deeply staining osteoblasts. The bone in the central area of the septum presented numerous, prominent and irregularly arranged incremental and resorption lines.

A comparable bone response with a somewhat different pattern was observed

Fig. 4. Cortisone injected animal. Detailed section of Fig. 3 showing the interdental septum and periodontal membrane. The deeply staining distal (left) and mesial (right) surfaces of the interdental septum are devoid of any appreciable bone activity. The crest presents a flattened, indented margin. Note the peculiar grouping and sharply outlined ovoid lacunar spaces. Note the irregularly indented margin of the medullary space in the central portion of the septum and the evidence of endosteal bone apposition bordering the medullary space in the lower region of the septum. With the exception of isolated areas, the periodontal membrane fibers are replaced by a homogeneous minutely fibrillar stroma. The fibroblasts are fewer in number and less regular in appearance than in the control. (Compare with Fig. 2) (H.E. x 290)

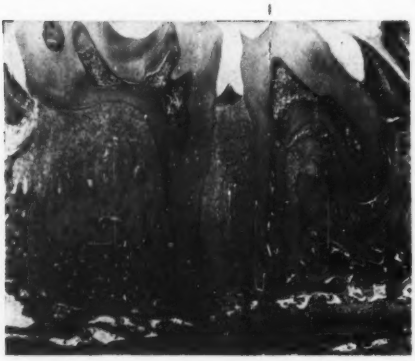

Fig. 5. Cortisone-Estrogen animal. Mesio-distal survey section through the mandible, showing the first and second molars and alveolar bone. (H.E. x 56)

in other animals in the cortisone-estrogen group. In such cases the crest of the septum and the distal surface consisted of newly formed bone containing ovoid osteocytes (Fig. 7 and 8). Numerous collagenous fibers were embedded in the newly formed bone and extended into the periodontal membrane. A beadlike layer of osteoblasts was in apposition with the bone margin at the crest and along the distal surface of the septum. In some areas the osteoblasts were partially embedded in pale staining osteoid.

The remainder of the septum was demarcated from the newly formed bone by an irregularly indented deeply staining appositional line. The bone in the remainder of the septum contained a relatively greater number of osteocytes in irregularly arranged, closely packed, irregularly shaped lacunae. The mesial surface of the septum was marked by contiguous slight indentations outlined by a fuzzy, deeply staining border, which in some instances contained multinucleated osteoclasts.

The changes in the bone in both the cortisone injected animals and the cortisone-estrogen animals were more notable in the interdental septa than in the bifurcation areas.

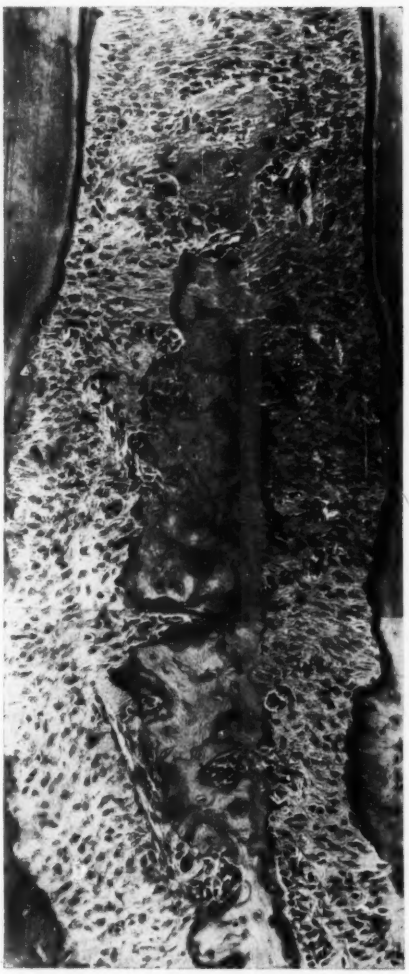

Fig. 6. Cortisone-Estrogen animal. Detailed section of Fig. 5 showing the interdental septum and periodontal membrane. At the crest there is an area of new bone formation with a bordering multilayered zone of fibroblasts, with osteoblasts partially imbedded in the bone. The mesial (left) and distal (right) surfaces consist for the most part of irregularly outlined indentations with deeply staining margins. In some areas along the distal surface of the septum and along the vessel channels, bead-like accumulations of osteoblasts are seen. (H.E. x 290)

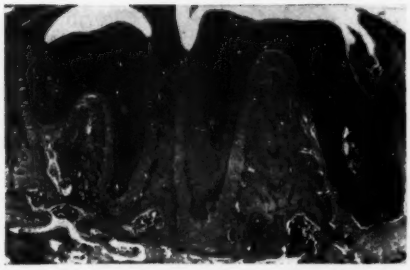

Fig. 7. Cortisone-Estrogen animal. Mesio-distal survey section through the mandible showing the first and second molars and alveolar bone. (H.E. x 56)

Fig. 8. Cortisone-Estrogen animal. Detailed section of Fig. 7 showing the interdental septum and periodontal membrane. Newly formed bone at the crest and along the distal surface (left) is demarcated from the underlying remainder of the septum by an irregular deeply staining appositional line. Note the comparative abundance of osteocytes in the latter portion of the septum and the marked contrast between the mesial (right) and distal septal surfaces. The periodontal membrane consists of numerous, densely arranged, large fibroblasts. The periodontal fibers are thin and strandlike in appearance. In some areas it is difficult to note a demarcation of these strands into separate fibers or fiber bundles. (H.E. x 290)

Bifurcation Area—The Interradicular Alveolar Bone

CONTROL ANIMALS

The margin of the mesial aspect of the alveolar bone in the bifurcation consisted of contiguous slight indentations (Fig. 9). Along the crest and distal surface there was evidence of new bone formation which in some areas was demarcated from the underlying bone by a clear cut appositional line. The individual medullary spaces were outlined in part by a layer of new bone and in part by an irregularly indented border.

CORTISONE INJECTED ANIMALS

Along both the mesial and distal aspects, the border of the bone was formed by a deeply staining margin devoid of any appreciable bone formation (Fig. 10). The bone margins bordering the medullary spaces stained deeply and with isolated exception presented no new bone formation.

CORTISONE-ESTROGEN ANIMALS

The alveolar bone in the bifurcation area differed from the cortisone injected animals and presented evidence of newly formed bone and osteoblasts in relation to segments of the distal surface as well as the crest (Fig. 11).

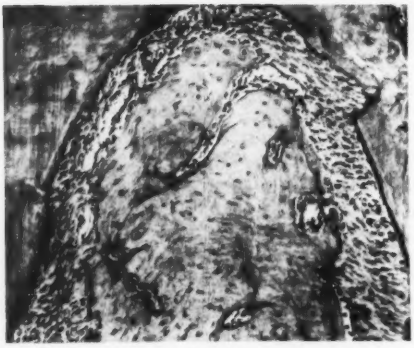

Fig. 9. Control animal. Alveolar bone in the bifurcation area of the first molar. The mesial margin (left) consists of contiguous slight indentations. There is some evidence of new bone formation at the crest and along the distal aspect. (H.E. x 215)

PERIODONTAL MEMBRANE

Control Animals

The periodontal membrane in relation to the interdental septa and bone in the bifurcation areas consisted of well formed bundles of collagen and numerous fibroblasts (Fig. 2 and 9). The nuclei of the fibroblasts varied in shape from ovoid to flattened, with intermediate elliptically shaped cells predominant. In many areas, the collagen fibers of the periodontal membrane blended imperceptibly with the osteoid margin of the adjacent bone.

Cortisone Injected Animals

The outlines of the periodontal membrane fibers were less clearly demarcated than in the control animals (Fig. 4 and 10). The fiber bundle pattern was replaced by a homogeneous fibrillar stroma which in some areas appeared granular. The fibroblasts were notably reduced in number and presented considerable variation in shape and irregularity in arrangement. Many of the nuclei appeared shrunken and hyperchromic.

Cortisone-Estrogen Animals

The most striking feature of the periodontal membrane was the large number and dense distribution of the fibroblasts and the prominent osteoblasts bordering the bone of the septum (Fig. 6, 8 and 11). Except for the crestal area, the nuclei of the fibroblasts were larger than those of the cortisone injected and the control animals. The collagen fibers appeared as thin strands which were numerous but not as clearly arranged in bundles as in the control animals.

CEMENTUM

The cementum in the experimental animals differed from the control animals. The differences were slight and more apparent in the apical third of the root. The thin peripheral zone of new cementum and

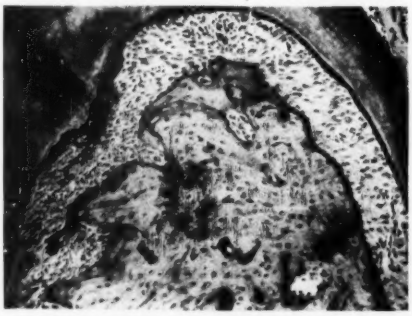

Fig. 10. Cortisone injected animal. Alveolar bone in the bifurcation area of the first molar. The entire margin of the bone adjacent to the periodontal membrane is devoid if new bone formation. (H.E. x 215)

adjacent cementoblasts seen in the control animals was diminished or completely absent in the cortisone injected animals. The cortisone-estrogen animals present some evidence of new cementum formation, which was not as extensive or generally distributed as it was in the control animals.

DISCUSSION

In evaluating changes in the surfaces of the alveolar bone adjacent to the periodontal membrane, consideration must be given to the pattern of bone formative and

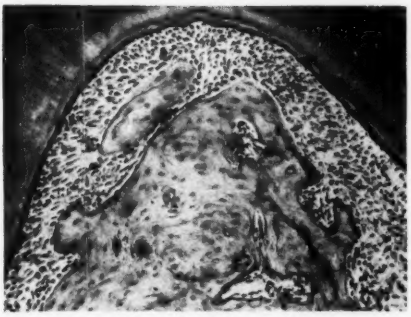

Fig. 11. Cortisone-Estrogen animal. Alveolar bone in the bifurcation area of the first molar. Note the prominent bead-like layer of osteoblasts with osteoid and newly formed bone in relation to a portion of the distal (right) surface as well as the crest and mesial surface. Also note the section of newly formed bone at the crest which is clearly demarcated from the underlying septum. (H.E. x 215)

bone resorptive activity normally present in this location. In the mouse, the molars erupt in a disto-occlusal direction. The mesial surface of the interdental septa and interradicular alveolar bone normally presents lacunar resorption with a scattering of multinuclear osteoclasts. Along the distal surface and the crest there is new bone formation with a border of osteoid and a bead-like arrangement of osteoblasts. When the molars have erupted so that they occlude with their antagonists, the intensity of the bone formative and bone resorptive activity is diminished, but the pattern remains the same.

In the young adult mice, (approximately six weeks) used in this study, the molars are erupted so that they occlude with their antagonists. In deference to the uncontrollable variable in the degree of bone formative and bone resorptive activity which exists in the alveolar bone in relation to teeth in different stages of eruption, our comparison of the different groups was based upon the bone in the region of the first molar.

Our findings corroborate the earlier report[1] that repeated injections with cortisone result in osteoporosis of alveolar bone. The daily dosage of cortisone used in the present study (0.25 mg.) was less than that employed previously (0.5 mg.), and the present findings were therefore somewhat less severe. However, even with the lesser dosage, the cortisone injected animals presented pronounced changes in the periodontal membrane surfaces of the alveolar bone in relation to all the teeth. The absence of any appreciable bone formative activity resulted in reduction of the height and width of the interdental and interradicular septa.

In the animals which received weekly injections of estradiol benzoate (600 R.U.) in addition to daily injections of cortisone (0.25 mg.) the microscopic appearance of the alveolar bone differed markedly from that seen in the animals injected with cortisone alone. The interdental and interradicular septa were no longer devoid of new bone formation. Instead newly formed osteoid and an adjacent layer of osteoblasts were distributed in conformity with the normal bone formative pattern. In many instances the interdental septa were clearly divisible into two zones, a crestal zone of regularly formed lamellated bone and an underlying zone marked by irregularly arranged deeply staining incremental lines and irregularly arranged groups of osteocytes. The resultant effect was an increase in the height and width of the interdental and interradicular septa in the direction of normal and reestablishment of the normal width of the periodontal membrane.

It could be interpreted, based upon the differences in the definitive zones which comprised the interdental septa, that the administration of estrogen along with cortisone led to a reversal of the osteoporotic tendency caused by cortisone alone. The estrogen, by stimulating bone formation along the periodontal membrane surfaces of the alveolar bone in relation to all the teeth effected a restoration of the height and width of the interdental septa by the addition of bone at the crest and along the distal surfaces.

The findings in the periodontal membrane provided further evidence that the

introduction of estrogen effected a modification in the changes induced by repeated injections of cortisone alone. In the animals receiving cortisone alone, there was a notable reduction in the number of fibroblasts in the periodontal membrane and a distortion in the appearance and distribution of the fibroblasts. In the animals receiving both cortisone and estrogen the fibroblasts were densely distributed throughout the periodontal membrane and were uniformly well formed with large, prominent nuclei.

The pronounced difference in the alveolar bone of the animals injected with cortisone alone, and those receiving both estrogen and cortisone warrants further analysis. Previous studies in mice are in agreement regarding the stimulation of endosteal bone formation[2-7] by systemic administration of estrogenic hormone. Two of these reports specifically mention that the pattern of apposition and resorption at the periodontal surface of the alveolar bone,[6] and the lamellated bone of the interradicular septa[7] are not affected by estrogen. It is noteworthy that our findings indicate changes in the periodontal membrane surface of alveolar bone when estrogen is administered to cortisone injected animals which differ from those described in previous studies in response to estrogen alone. In cortisone injected animals, the stimulating effect of estrogen upon bone formation was most marked along the periodontal membrane surfaces of the alveolar bone. These are areas described as unaffected when estrogen is administered alone. It was observed that in the animals receiving both estrogen and cortisone the extent and distribution of endosteal bone formation were more marked than in the animals receiving cortisone alone. However, the difference between the two groups of animals in this regard was far less striking than the differences seen along the periodontal membrane surfaces of the bone.

The osteogenic effect of estrogen upon the periodontal membrane surfaces of alveolar bone, when it is administered to cortisone-injected animals, may be interpreted as an attempt to compensate for the anti-anabolic effect of the cortisone upon the bone. This would represent an effort to re-establish the physiologic anabolic-catabolic equilibrium of the bone economy.

It has been suggested[8] that the osteoporosis induced by cortisone is essentially a manifestation of the anti-anabolic effect of the drug. The normal bone formative activity along the periodontal membrane surface of the alveolar bone is seriously impaired in cortisone injected animals. Under the conditions of the present experiment, estrogen is introduced in animals with an existent hormonal imbalance. The estrogen stimulates bone formation in areas of the alveolar bone where the detrimental effect of the existent hormonal imbalance is manifest, despite the fact that these are areas reported as unaffected by the administration of estrogen alone.

The present study represents the first demonstration in animals of the restoration of previously destroyed alveolar bone by the introduction of a systemically administered drug. It is difficult to disregard the possible implications of these findings in relation to the future of periodontal practice. However, conjecture in this connection must be prefaced by the realization that our findings are in experimental animals, selected because of their peculiar suitability for this type of investigation.

Attractive though the prospect may be, no inference may be drawn from our findings regarding the feasibility of systemic therapy for the restoration of tooth supporting bone in humans with chronic destructive periodontal disease. Estrogens have been administered in humans for the correction of osteoporotic defects in bone [9, 10, 11]. In the present study, estradiol benzoate was used as an "experimental tool" to investigate the validity of a principle. It should not be inferred that estrogenic hormone is the only substance with which the restoration of alveolar bone may be attained in animals, nor may it be presumed that comparable changes would occur in

humans. The presentation of our findings at this early stage in our investigations is intended primarily as a stimulus for additional research in a similar direction.

CONCLUSION

The systemic administration of estradiol benzoate in young adult white mice receiving daily injections with cortisone effected a modification of the osteoporotic effect observed when cortisone is administered alone. The systemic administration of estrogen resulted in the stimulation of bone formative activity which effected a restoration in the height and width of the alveolar bone.

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TEMPLE UNIVERSITY ANNOUNCES A COURSE IN CLINICAL OCCLUSION

By Dr. Lewis Fox, South Norwalk, Conn.

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Subgingival Curettage

by BERNARD S. CHAIKIN,* *Boston, Massachusetts*

THE objectives in the therapy of any disease process comprise not only the restoration to health of the involved tissues but also of a return to a normal physiology. In the case of periodontal therapy, these aims consist of a "normal" gingival attachment and a radiographic intact lamina dura and crestal bone. In order to achieve these results the periodontist as a rule must eliminate pocket formation. One method is subgingival curettage, a procedure to be discussed in this paper.

RATIONALE

Periodontal pockets may be formed as a result of a hyperplasia of the gingival tissue increasing the depth of the retracted tissue or may occur because of apical proliferation and subsequent detachment of the epithelial attachment. The relationship of the base of the pocket to the alveolar crest serves as a basis for classification of periodontal pockets. Those whose base on the tooth lies coronal to the alveolar crest have been classified as supra-bony while those apical to this reference location are termed infra-bony. Pockets are also classified as shallow (less than 3 mm. in depth) or deep (over this measurement). The symptoms of pocket formation constitute not only a retraction of the gingival tissue from the tooth to a certain depth but also an inflammatory process in the gingival tissue. Discoloration, bleeding, exudation and calculus are the essential features of pocket formation. Calculus attached to the tooth surface and an ulcerated sulcular epithelium lining the pocket with a marked inflammatory infiltrate in the gingival corium, are the main findings of histological examination.

Pockets from a topographical point of view are located on one or more surfaces of the tooth. Their size, shape and contour vary from shallow "U" formations to a deep tortuous, spiralling topography.

The objective of subgingival curettage is the conversion of a periodontal pocket into a healthy gingival sulcus, characterized by a pink, firm, well adhering gingival margin, the topography of the gingival tissue being physiologic in nature. Thus for subgingival curettage those pockets which can be so converted are selected. Closely identified with subgingival curettage is expected reattachment. However, it must be emphasized that this procedure can also be performed in those instances where pocket elimination will be achieved by shrinkage of the gingival tissue, decreasing the depth of the retracted gingival wall and healing of the sulcular epithelium.

It has repeatedly been demonstrated that removal of calculus from the tooth surface with which periodontal pockets are associated, in many instances results in the healing of the gingival tissue eliminating the pocket. For the most part, however, especially in deep pockets, but not necessarily so, this healing does not occur. Therefore, after scaling, the operator must reexamine to determine the status of health of the gingival attachment. All pockets which have persisted should be charted as to location, type, size, and contour. Subgingival curettage is employed only for those pockets for which therapy is indicated. Shallow supra-bony pockets, the gingival tissue being soft, red, and edematous, respond beneficially; those pockets where because of location and local environment, reattachment may occur, are also treated. Infra-bony pockets fall into this latter category.

In the curettage procedure two features of the technique must be discussed. First the removal of the sulcular epithelium and second, that of the elimination of the epithelial attachment. In the microscopic

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Presented at the American Academy of Periodontology, Cleveland, Ohio, September 26, 1953.

Fig. 1. Periodontitis
Upper. Before treatment, supra-bony pockets were 5 to 6 mm. in depth. The gingivae were edematous, inflamed and could be lifted away from many teeth.
Lower. After subgingival curettage. The sulcus depth was 1 mm. or less due to shrinkage of the gingivae in the healing process.

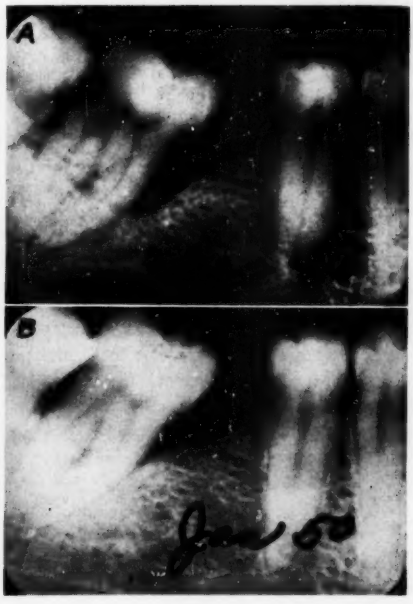

Figure 2
Upper. Deep infra-bony pocket extending around the lingual and mesial surfaces of the lower second molar.
Lower. Three years after subgingival curettage. There was an apparent alveolar bone regeneration.

study of the periodontal pocket, it has been demonstrated that the sulcular epithelium not only shows evidence of ulceration but also of proliferation into the gingival corium. As in any other debridement technique, the covering epthelium (sulcular) is eliminated allowing a new sulcular epithelium to grow over the wound. As in the healing of an ulcer elsewhere, epithelium from the lateral surfaces will proliferate and cover the defect. The problem of the elimination of the epithelial attachment resolves itself into the fact as to whether or not reattachment is expected. Where no reattachment is sought after, no effort is made to remove this tissue. In those pockets where reattachment is expected, this attachment must be eliminated if connective tissue fibers are to become incorporated into a new layer of cementum produced on the tooth surface. There are many contraindications for treatment by subgingival curettage, but these will not be considered here.

TECHNIQUE

In the management of the shallow supra-bony pocket, the sulcular epithelium is curetted; any subgingival calculus that may be present is removed, the expectation being that shrinkage of the gingivae will result producing a sulcus and a healthy gingival tissue within normal limits. For the deep supra-bony pockets, the gingivae should be anesthetized by a topical or infiltration anaesthetic and the area to be operated upon isolated with cotton rolls or gauze. With small curets having very sharp edges, the tooth side of the pocket is scaled of every vestige of calcarious deposits and necrotic cementum. The operator should be mindful of the tooth anatomy which may present a

depression or concavity on the mesial or distal surface of the root, attached cementicles should not be mistaken for calcarious deposits, likewise the bulge of the enamel at the cemento-enamel junction should not be mistaken for calculus. When the tooth side of the pocket has been thoroughly scaled, it should feel smooth. Air may be blown into the pocket to separate the soft tissue from the tooth for better inspection to determine whether all deposits have been removed.

After this step in the therapeutic procedure has been accomplished, attention is next focused upon the soft tissue side of the pocket. The inner portion of the retracted gingival wall is curetted removing the inflamed gingival tissue including the sulcular epithelium and also approximately 1 mm. of the crestal gingival tissue. This operative procedure is accomplished by directing the cutting edge of the curet towards the soft tissue. With resolution of the inflammation and healing of the sulcular epithelium, shrinkage of the gingival tissue will result.

In the infra-bony type of pocket where reattachment is attempted, the epithelial attachment is removed in order to prevent it from acting as a barrier against any possible regrowth of the periodontal membrane. Therefore, after the root surface has been scaled and the soft tissue side of the pocket has been curetted, a sharp pointed curet is used to remove the epithelial attachment in its entirety.

In order to maintain an undisturbed blood clot in the pocket, a rectangular strip of adhesive tin foil is placed interproximally over the operated area. This is covered with a surgical pack and in the posterior segments, a second larger piece of tin foil is added to cover the entire pack. The latter is not used in the anterior segments because of its esthetics and if the patient is cautioned against disturbing the pack, it will remain intact for the prescribed length of time of one week when it is removed. The patient is warned against massage for another week to allow for further cellular organization, after which the usual oral physio-therapy instructions are carried out.

Supra-bony pockets, treated by curettage will heal in one of four different ways, which are:

(1) By shrinkage of the edematous, inflamed gingivae to the point where the sulcus is of negligible depth (Fig. 1).

(2) By circumferential shrinkage in deep pockets where for all practical purposes, the sulcus has little depth. Actually though, the gingiva has tightened around the tooth to such a degree that a negligible sulcus to the probe results, but there is no physiologic union between the tooth and soft tissue. Thus the latter can be forcibly separated from the tooth. As long as this relationship exists intact, it will continue to be healthy, but food and debris might injure the gingivae by mechanical means thus initiating an inflammatory process and cause the sulcus to open up which may create an environment that might be instrumental in deepening the pocket.

(3) Healing may take place by the regeneration of the periodontium to a higher crownward level than before treatment (Fig. 2). This has been shown clinically and may indicate the reattachment of a regenerated periodontal membrane. These results are best seen in the infra-bony pockets, and the narrower the pocket, the better will be the result.

(4) Healing may also take place by the resolution of the inflammation but still maintaining a deepened crevice which may continue to break down and get deeper. This type of pocket would best be treated by gingivectomy.

SUMMARY

Supra-bony pockets treated by subgingival curettage will heal in most instances by shrinkage of the overlying soft tissue, although in many cases, regeneration of the periodontium will take place. On the other hand the infra-bony pocket will heal by a combination of shrinkage of the soft tissue, and regeneration of the bone, the degree being dependent upon its width.

Union of the Enamel and Gingival Epithelium*

by Luz C. Macapanpan,**Chicago

THE manner of attachment of the epithelium to the enamel has long been debated. Prior to the publication by Gottlieb in 1923, the theory prevailed that no organic connection existed between tooth and epithelium, but merely an apposition of the gingiva to the enamel surface due to tissue tone and elasticity of the connective tissue of the gingiva.

Gottlieb, who had himself thought at first that the epithelium simply "hugged" the enamel, disproved that concept with microscopic evidence. He showed that in the unerupted tooth, after the formation of enamel, the united enamel epithelium regresses to a stratified epithelium which remains organically attached to the enamel. From the moment the tooth erupts, the reduced enamel epithelium unites with the oral epithelium and is thereafter termed the epithelial attachment.

Bodecker and Applebaum ('34) showed in specially prepared ground sections of teeth and supporting tissues that the relations between enamel and epithelium remained undisturbed. Turner ('27) and Kronfeld ('30) observed the adherence of epithelium to extracted teeth.

Since then, other observations and experiments have strengthened this view. Toller ('40) froze and sectioned the teeth and surrounding tissues in young dogs and pulled away the free margin of the gingiva from the tooth. It was shown that after a certain depth at which the epithelium peeled off cleanly from the enamel surface, the epithelial attachment tore and left part of the epithelial attachment on the enamel surface. This demonstrated the strong attachment of the epithelium to the enamel.

This stand has been challenged by Waerhaug ('52), who based his conclusions on experiments on both animal and human material. He inserted blunt, but extremely thin steel blades into the gingival sulcus until resistance was felt and found consistently that resistance was offered at the level of the cemento-enamel junction in spite of the very minimal force used (0.1 gram). In these cases, he observed that the epithelial attachment was histologically normal. In those instances in which some epithelial cells remained on the tooth surface after the gingiva had been pulled away, he found that a tiny jet of water was sufficient to wash them off. Waerhaug believes that these cells are merely degenerated epithelial cells which could not be

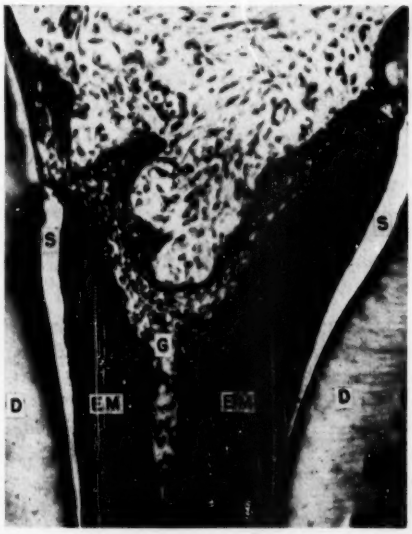

Fig. 1. Photomicrograph of a mesio-distal section through the interdental space between the upper right first and second rat molars showing the union between enamel and gingival epithelium. Magnification: X 270.

*This investigation was carried out under contract No. W-49-007 MD-496 with the Medical Research and Development Board, Office of the Surgeon General of the United States Army and partially supported by the Dental Protective Association of the United States.

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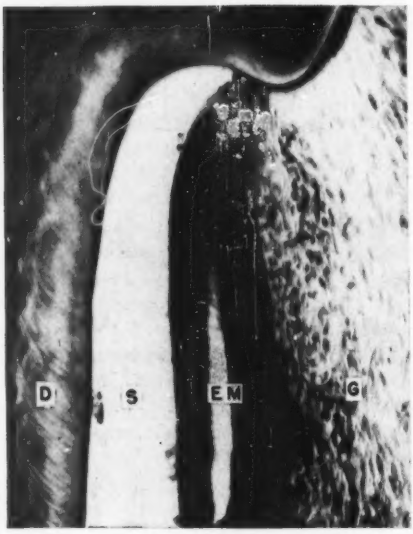

Fig. 2. Photomicrograph of a mesio-distal section through the distal side of an upper right third molar showing the union between enamel and gingival epithelium. Magnification: X 270. EM—Enamel matrix. S—Shrinkage space. D—Dentin. G—Gingival epithelium.

easily desquamated because of their peculiar position. He further noted that epithelial cells can be found attached to the surface of artificial crowns which have been in contact with the gingiva, and that their strength and appearance was very similar to that of normal "epithelial attachment." Because of these findings, Waerhaug denies the existence of an organic union between epithelium and enamel and suggests that the term "epithelial cuff" be substituted for "epithelial attachment."

Until unequivocal histologic and experimental evidence is obtained, the question of the mode of attachment cannot be decided, but to deny the existence of an epithelial attachment simply because we do not know the mechanism of attachment is unwarranted. However, it seems necessary to evaluate any chance findings in this area.

Two observations which seem to support the concept of an organic union between enamel and epithelium will be presented: (1) on a rat molar, and (2) on a human tooth in which the enamel matrix has been preserved along with the supporting tissues of the tooth. Such findings are neither rare nor isolated. They have been observed many times in our own rat material and in human material.*

The relations of epithelium and enamel were studied on mesio-distal sections through the interdental space between the upper right first and second molars of an adult albino rat (Fig. 1) and through the distal side of an upper right third molar

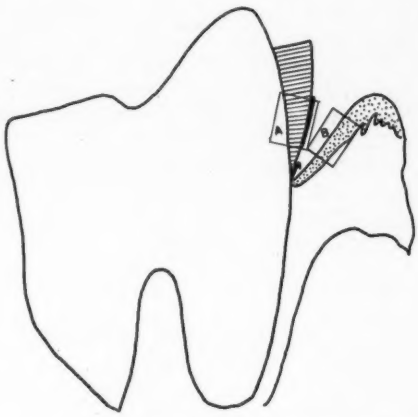

Fig. 3. Diagram of a bucco-lingual section through the human molar of Figs. 4 and 5. The areas shown in Figs. 4 and 5 are indicated by squares.

in another rat (Fig. 2). Almost the entire thickness of the enamel matrix (EM) at the cervical half of the teeth has been preserved and can be followed to the cemento-enamel junction. During routine histologic procedures, shrinkage of the tissue occurred. It is interesting to note that the enamel matrix (EM) remained attached to the gingival papilla (G) but had shrunk away from the dentin (D) at certain areas (S).

In all cases observed, enamel remained

*Personal communication, V. G. Villa.

Fig. 4. Photomicrograph or area A in Fig. 3. EM—enamel matrix. D—dentin. Note the layer of epithelial cells (C) attached to the surface of the enamel matrix. Magnification: X 270.

Fig. 5. Photomicrograph of area B in Fig. 3 showing the gingiva that was pulled away from the tooth surface. The surface of the epithelium (C) is denuded of superficial cells. Magnification: X 270.

attached to epithelium but had shrunk away from the dentin. These findings evidently tend to show that the connection between enamel and dentin was weaker than that between enamel and epithelium.

The second case is that of a human molar in which by an artefact during preparation, the gingiva had been pulled away from the enamel (Figs. 3, 4, 5). On the enamel matrix, a layer of cells has remained in seemingly organic union with the enamel (Fig. 4). The gingival epithelium (Fig. 5) is denuded of its superficial cells. The tear, therefore, occurred not between enamel and epithelium but was *in* the epithelium, leaving a portion of the epithelium still attached to the enamel matrix.

SUMMARY

Two chance findings are presented which may throw light on the question of the existence of an epithelial attachment. One case (rat molar) showed the preserved enamel matrix still attached to epithelium but shrunken away from the dentin, thus demonstrating a stronger attachment between enamel and epithelium than between enamel and dentin. The other case (human molar) showed a layer of epithelial cells still attached to the enamel surface even after the gingiva had been pulled away, indicating that where the gingiva is detached from the enamel, the tear occurs *in* the epithelium. Again, this points to the firmer union between enamel and epithelium.

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Gingivectomy and Inter-Dental Resection

by RICHARD CHACE, D.D.S., *Orlando, Florida*

GUM resection should not be considered as a thing apart from subgingival curettage. It should merely be thought of as a method of producing soft tissue atrophy quickly and surely. A properly executed gum resection should differ from sub-gingival curettage in one respect only. In the former the gingival wall of the pocket is surgically removed. It must be understood that skillful curettage will produce atrophy of the gingivae, particularly when packing is employed. In the shallow or moderate pocket, this is often the method of choice. If the pocket is deep, however, if the gingival wall becomes fibrous, if gingival hypertrophy is great, or if granulomatous tissue dominates the deeper part of the gingival wall, the gum resection becomes a more practical procedure.

It has been said that a gum resection is a radical operation, and indeed, it can be one if the operator lacks proper skill and judgment. Only needless destruction of tissue is radical. The conservative periodontist must make every effort to produce a new connective tissue attachment whenever he has a reasonable chance of succeeding, and tissue conservation must be his watchword.

It should be axiomatic that no definitive treatment of the periodontal pocket should be made without preliminary treatment. This consists of preliminary scaling and curettage, correction of major occlusal abnormalities, and the teaching of toothbrushing and interdental stimulation. The response to this preliminary treatment will give the operator valuable information on how well the individual patient responds to therapy. Sometimes an exceptionally favorable response will cause the dentist to continue with curettage even in the presence of deep pockts. Sometimes slight to moderate pockets will improve so little in the preliminary treatment that resection will be decided on. In the latter case, however, the experienced periodontist will carefully re-examine the etiological factors before going further into treatment.

Gum resection may often be used in conjunction with and supplemental to subgingival curettage at the same sitting. Despite careful charting, individual pockets often prove deeper than they appear on first examination. Such pockets are curetted carefully and their exact form determined. If soft tissue atrophy appears unlikely in such areas the gingival wall can be excised and a pack placed. The mesio-lingual surface on the upper first molar is a common site for this simple supplementary resection. Since definitive curettage is often done under local anesthesia, the extra time and trouble involved is negligible. Such minor resections usually heal very quickly and require only a few days of wearing the periodontal pack.

MODIFIED RESECTION TECHNIQUE

Scattered, individual, deep pockets may be treated by modified resection without doing a gingivectomy over the whole mouth. Such pockets should receive special attention during the preliminary treatment so that all gingival inflammation is resolved. In such pockets at least partial closure by reattachment is attempted. Much has been made over the difficulty of gaining reattachment because of the chronic inflammation present. By thoroughly removing all calculus, by curetting the granulation tissue, by medication, and by correcting the occlusion several weeks before the definitive treatment, the percentage and the degree of new connective tissue reattachment can be greatly increased. It must be remembered that reattachment is often only partially successful and that recurettement and sealing of the pocket under anesthesia may be desirable later. Sometimes several curettements are necessary to

obtain maximum closure. These are done at recall appointments and usually require only a few minutes. Here again we see how the gum resection and sub-gingival curettage complement each other.

The individual pockets that are treated by modified resections and when a new connective tissue attachment is attempted are of three types:

1. Inter-proximal, supra-bony pockets when there are no labial or lingual pockets.
2. Infra-bony pockets.
3. Narrow, deep, labial pockets.

Aside from the special care that must be taken during the preliminary treatment, these three types have one thing in common. In the definitive treatment of all of them, the epithelial attachment must be removed. Orban[1] has recently illustrated the difficulty of clinically determining how far apically one must curet in order to remove this epithelial attachment. From the standpoint of the clinician it can be said that a sharp, fine curet is used to remove the soft tissue attachment down to tissue that feels firmly attached to the rest. From a practical point of view this procedure is generally successful.

THE INTERDENTAL RESECTION

Deep, interproximal pockets without buccal or lingual involvements are quite common. If they are treated by simple excision, healing results in a punched-out interdental space that not only has poor esthetics but catches food. Beube's[2] interdental resection is an effective method of eliminating this type of pocket. While we are discussing the elimination of individual pockets by modified resection techniques, it may be noted that this and the other special techniques may be incorporated into the regular gingivectomy.

The incision is made with a Towner 19 or 20 scalpel at a 45 degree angle, about 3 mm from the crest of the papilla and is carried apically until the bone is reached. A similar incision is made on the other side of the papilla and a wedge-shaped piece of tissue is removed. Any tabs of tissue or any granulomatous tissue is removed. The root surfaces are then carefully planned until they are quite glassy. The area is then isolated from saliva and while a blood clot is forming in the crater area the all important gingival bevel is obtained by scraping the wound margins with a very sharp Kirkland or Buck scalpel. This shaping and beveling not only gives the operated area the proper architecture but it removes epithelial tissue from the spot where we want it least. Beveling may also be done by excising with a very sharp knife or by using the electro-cautery, but the scraping method is both sure and easy.

By the time beveling is complete, the crater of the operation has a well formed blood clot which must not be displaced in the placing of the pack. A softer pack is used than in the gingivectomy and care must be taken that the beveled edges are covered. Adhesive tinfoil is helpful in isolating the operated area. The pack should be changed several times in the two week period of healing. Superoxol used at pack changes is an aid in healing. When the pack is removed the patient must be instructed to resume gentle interdental stimulation.

THE INFRA-BONY POCKET

The treatment of the infra-bony pocket varies considerably with its shape and location. A narrow, deep, interproximal pocket often responds to a thorough curettage under local anesthesia. A broader pocket requires that the papilla be resected before the curettage is started. This incision, made with a Towner or Sanders scalpel, also removes any buccal or lingual extensions of the pocket that are supra-bony. The root surface must be curetted until it is perfectly smooth and all tabs of granulation tissue must be removed. A blood clot is allowed to fill the infra-bony space and the edge of the buccal and lingual incision is carefully scraped with a sharp Buck scalpel to produce the bevel necessary for good

gingival architecture. Care must be taken not to force the pack into the infra-bony space.

The less the distance is from the crest of the bone to the root surface, the greater the chance is for bony regeneration. When the distance is great, it is generally more successful to place the pack almost to the depth of the pocket and allow the crater to granulate up from the bottom. The pack is kept on for at least three weeks and is changed four or five times.

NARROW SUPRA-BONY POCKETS

Narrow, deep, supra-bony pockets often occur on the labial surfaces of both anterior and posterior teeth. Their complete excision not only results in a poor esthetic result but often carries the gingival margin down into the soft, alveolar tissue. Such pockets are best treated by a modified resection. About two or three mm of the gingival margin is excised with a Kirkland scalpel. The pocket is then carefully curetted. The tissue is pressed against the tooth with a sterile gauze pad until a firm clot is formed and a pack is placed. This type of pocket is almost always closed by this procedure although it is occasionally necessary to re-curet under anesthesia at a later date.

THE STANDARD GUM RESECTION

The gum resection comes into its own as a method of eliminating the horizontal pockets of moderate to severe periodontitis simplex. The Kirkland[3] operation is the best known and most widely used and it may be modified, when indicated, with any of the special resection techniques mentioned above. In this type of operation, the gingival pocket is eliminated to the epithelial attachment being careful to bevel the incision so that shelf formation will be minimized. It is often possible to escallop the labial incision around the anterior teeth and produce a better esthetic result. After the gingival tissue is removed the area should be carefully probed to see if any supra-bony pocket tissue was missed. Pocket outline may be quite irregular and portions of the gingival wall are often inadvertently left. Sometimes a pocket may be discovered to be partly infra-bony and, if so, will present an opportunity for reattachment. Quite frequently it will be necessary to recontour the remaining gingival tissue with sharp scissors or additional excision with a sharp Sanders scalpel. Very little calculus should remain on the root surfaces if proper preliminary treatment has been done. Any that is left will be easily removed in the routine smoothing of all root surfaces. While curets may be used for this step, it is accomplished more easily by using the 2K, 3K, and 13K instruments. A pushing stroke is employed, working labio-lingually as well as apically. All softened root surface must be removed. After a glassy smooth root surface on all teeth has been obtained, the area is carefully dried and rechecked with fine sharp curets. All remaining tabs of granulation tissue are removed and the final gingival bevel is obtained by scraping. A pack of thick, putty-like consistency is applied. It is changed in one week and after another week is removed. Sensitivity of root surfaces is usually transitory. The patient is able to use a soft brush immediately.

Bifurcation and trifurcation involvements may be successfully treated by gum resection if other factors are favorable.[4] Occasionally the deeper aspects of these pockets that involve bifurcations are infra-bony and are, therefore, eligible for an attempt at reattachment. More frequently it will be found that careful contouring of the gum tissue will produce an area that can be kept clean with that efficient watchdog of the periodontist—the round toothpick.

VERY DEEP POCKETS

Contrary to popular opinion, the standard gum resection is not very useful in the very advanced periodontal case. All of us are called on to treat apparently hopeless mouths. While this should not be a routine

procedure, it is sometimes preferable to full-mouth extraction. In such cases a partial resection gives good results. No attempt is made to resect the pocket to its base. Instead, the incision is made with the idea of eliminating as much of the pocket as possible and still to have acceptable gingival architecture. The remaining pocket tissue is curetted very carefully and the lining of the soft tissue wall is removed. While this procedure is far from ideal, it is amazing how well the mouth looks after treatment. In these cases the patient should be taught to portpolish his own teeth as part of his daily home care regime.

It will probably be noted how often the smoothing of the root surfaces has been referred to in this paper. Most descriptions of the gingivectomy technique relegate this step to a position of unimportance. It must be remembered, however, that gum regeneration follows every gingivectomy. Porosity of the root surface often constitutes enough of an irritant to encourage the new gingival tissue to hypertrophy and form new pockets. This happens far oftener than is generally supposed. An analogy between the effect of a smooth root surface and glazed porcelain on gum tissue may be made. The fact that exemplary home care may prevent pocket recurrence in the average case should not blind the operator to the importance of careful root curettage.

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Consideration of Tongue-thrusting as a Factor in Periodontal Disease

by HAROLD G. RAY, D.D.S., M.S.,* AND HAROLD A. SANTOS, D.D.S., B.S.,**
San Francisco, Calif.

THE multiplicity of etiologic factors in periodontal disease demands complete consideration of the entire gamut of influences brought about in the complete functioning of the masticatory mechanism. The role of local irritation, modified by systemic states, the actual occlusion and stresses borne by the teeth in the process of chewing, hereditary influences and developmental anomalies have all been extensively considered. The function of deglutition and the organs concerned with it and its effect upon the periodontium has to date not been adequately demonstrated. Obviously during the deglutition act, forces are exerted upon the teeth and the periodontium which can fall within the range of functional stimulus or extend to the level of injury. The forces that the tongue can exert in this act, if uncontrolled by compensating mechanisms, can modify the reaction of the periodontium to the other complicating factors in periodontal disease. We have become increasingly aware of the untoward influence that abnormal tongue habits might have upon progressive periodontal disease. As a result of casual observations in the clinic some striking cases precipitated our curiosity and upon more careful observation of general clinical materials we have become interested in this phase of function or dysfunction as it may be related to this problem.

REVIEW OF LITERATURE

The literature contains many references to the tongue in contributing to the production of open bite cases. Orthodontists and pedodontists have long known that the tongue can produce procumbency and arrest eruption.[1,2] However, the consideration of forces applied against the resistance of normal facial development has not been adequately considered in these writings. Breitner[3] points out that the forces of the tongue from within the dental arches is normally compensated by the action of the musculature of the cheeks and lips. The inference here is that as a result of macroglossia the tongue tends to displace the teeth labially, or to enlarge the dental arch as a result of the lack of resistance of the lips and cheeks. This is of course true, but often the macroglossia does not develop until the arches are completely formed, and this may have a tendency to displace the teeth even though they meet with proper resistance of the facial muscles. Physiologic discrepancies, such as a hyposensitive palate, can bring about an abnormal handling of the bolus of food in the process of deglutition to produce adverse effects. Sweet[4] has described, very well, that in these cases the tongue is thrust upward onto the hard palate and thrust forward against or between the anterior teeth in order to force the bolus of food into the pharyngeal region. The force of this abnormal thrust can certainly cause migration of the teeth or contribute to an open-bite. Miller corroborates this statement as quoted by Asnis.[5] Essentially a hyposensitive palate is the cardinal symptom of an abnormal thrust of the tongue, as a result of malfunction.

Teuscher[1] attributes many cases of acquired tongue thrust to a substitution for finger sucking. Tisdale[2] and Flesher[2] feel that the thrusting is a habit acquired during the mixed dentition when the tongue is placed into a space created by the loss of one or more deciduous teeth. This habit is more easily acquired when all of the deciduous anteriors are lost at about the

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same time and replacement by the permanent teeth is delayed. Whitman[6] and Sweet have independently stated that some patients develop the habit of tongue-thrusting as a result of using a bottle nipple which was too long or too free-flowing. In this instance there is no effort needed to nurse and therefore the child does not learn to raise the posterior part of his tongue. Patients in whom the gag-reflex is lacking or diminished have no experience in swallowing normally. Whitman[6] points out that some patients develop tongue perversions following tonsillectomy, probably due to injuries to the sensory nerves in the faucial area. Tongue habits can be projected into adult life so that the posterior portion of the tongue is never used to seal the oral cavity during deglutition.

In general, the literature reveals that an awareness of tongue habits and macroglossia in relation to malocclusion is evident, but the relationship between the abnormalities and periodontal disease does not seem to be clearly established. Sorrin[7] has cited a case of periodontal involvement of the anterior teeth as a result of this problem.

Sweet's reference to the possibility of hyposensitive uvulas, Whitman's and Miller's consideration of "hypoesthesia" of the soft palate seem to be the most pertinent references to the relationship of tongue thrusting and macroglossia we have examined.

The tongue in normal swallowing is in such a position that the dorsal surface is in contact with the hard and soft palates, exerting its pressure against them in an upward and lateral direction. Its tip and edges do not protrude against the teeth at any point. The tip of the tongue does not come forward any further than the lingual gingival margin of the maxillary incisors, and it does not rest on the incisor teeth.

In tongue-thrusting cases the tongue does not arch up in the palatal vault, but lies flat in the mouth. Due to this lack of arching, it needs more room laterally and therefore protrudes anteriorly causing the maxillary incisors to be pushed forward, upward and outward. In some cases the tongue extrudes laterally between the occlusal surfaces of the posterior teeth, and the open-bite is produced in one or both of the segments.

CLINICAL FINDINGS

Thirty-two cases with macroglossia and abnormal tongue-thrusting habits were examined in our general clinic, at the University of California College of Dentistry. All of these cases had periodontitis. The accompanying table Figure 1 shows the characteristic relationship of hyposensitive palate to tongue-thrusting, to macroglossia,

Case No.	*Age*	*Sex*	*I*	*II*	*III*	*IV*
# 1	40	F	—	X	X	—*
# 2	50	F	—	X	X	X*
# 3	57	M	X	X	X	X
# 4	28	M	X	X	X	X
# 5	36	F	X	X	X	X
# 6	27	M	X	X	X	X
# 7	58	M	X	X	X	X
# 8	37	F	X	X	X	X
# 9	46	F	X	X	X	X
#10	53	F	X	X	X	X
#11	26	F	X	X	X	X
#12	41	F	X	X	X	X
#13	43	M	X	X	X	X
#14	22	M	X	X	X	X
#15	46	F	—	X	X	X
#16	38	F	X	X	X	X
#17	65	F	X	X	X	X
#18	35	M	X	X	X	X
#19	28	F	X	X	X	X
#20	36	F	X	X	X	—
#21	41	M	X	X	X	X*
#22	29	F	X	X	X	X
#23	39	F	X	X	X	X
#24	34	M	X	X	X	X
#25	26	M	X	X	X	X
#26	32	F	X	X	X	X
#27	31	F	X	X	X	—
#28	37	M	X	X	X	X
#29	20	M	X	X	X	—*
#30	53	M	X	X	X	—*
#31	52	M	X	X	X	—*
#32	32	F	X	X	X	X

Fig. 1. Chart one lists case number, age, and sex of patient. Column I denotes hyposensitive palate, column II tongue-thrusting, Column III macroglossia and Column IV periodontal involvment of anterior teeth.

Key to chart: X denotes affected, — denotes not affected, * denotes open-bite cases when anterior teeth do not occlude in any excursion.

Fig. 2. Stimulation of uvula with mouth mirror does not excite normal gag-reflex.

and to periodontal involvement of the anterior teeth. For purposes of clarification the presence of a hyposensitive palate was determined by vigorous stimulation of the uvula, with a mouth mirror, without exciting the normal gag-reflex. We feel that the lack of sensitivity of the uvula forces the tongue to play an abnormal part in the involuntary swallowing act. The patients presenting this condition are designated by an "x" in the column marked "I." This condition is illustrated by Figure 2. The symptom of tongue-thrusting was determined by observation in which the patient thrust the tongue upward onto the anterior portion of the hard palate and then forward against or between the anterior teeth as part of the abnormal swallowing process. Thus the normal sequence of events, in the act of deglutition, is varied as a result of necessity and continues as an unconscious habit to the patient. An illustration of this characteristic is shown in Figure 3. The patients showing presence of this habit are designated with an "x" in column II of Figure 1. The presence of macroglossia was determined by observation. This symptom is due, we feel, to hypertrophy of the muscle fibers of the tongue as a result of excessive function. In some cases this is so pronounced and the tongue so completely fills the oral cavity in rest position that a scalloping results on the periphery. This is illustrated by Figure 4, and the patients presenting this feature are designated with an "x" in column III. The presence of periodontal involvement of the anterior teeth in these cases was determined by clinical observation and correlation with roentgenograms. This finding was particularly evident in the maxillary arch, with excessive involvement on the lingual of the incisors and extending to the first bicuspids. This is illustrated by the roentgenological picture in Figure 5, and the cases presenting this feature are designated by an "x" in column IV. The sequence and occurrence

Fig. 3. The tongue thrust forward against and between the anterior teeth in the act of deglutition.

of the foregoing characteristics are so regular that they might be considered a syndrome. This syndrome is much more common than the literature would indicate. These cases were observed in the natural course of clinical instruction and therefore, do not represent a screening of a large group of patients nor a concerted effort to uncover them.

In analyzing the findings illustrated in Table 1 it will be noted that the correlation between hyposensitive palates, tongue-thrusting and macroglossia is excellent. In only three cases of the total, macroglossia and tongue-thrusting existed without the association of the hyposensitive palate. It would seem therefore that in cases in which the uvula is lacking in sensitivity the habit of tongue-thrusting with accompanying macroglossia are predetermined sequela. The three cases in which tongue-thrusting and macroglossia were found to be present

without hyposensitivity of the palate can be explained by any of the other reasons for tongue habits such as finger sucking, etc., previously outlined. In addition, cases number 1 and 2 were open-bite cases which further substantiates the premise that the habit was prevalent during childhood.

In considering column IV it will be noted that all cases examined were cases of periodontal disease, particularly periodontitis. Column IV indicates that there is a high incidence of periodontal disease involving the anterior teeth associated with this tongue syndrome and of necessity has to be considered as part of the etiologic mechanism in these instances. Six of the cases presented did not show evidence of periodontal involvement of the anterior teeth. Four of these cases had open-bites; two had no periodontal involvement of the upper anteriors. The remainder, however, presented definite periodontal involvement of the upper anteriors in varying degrees of severity. Figure 6 is a table illustrating the group without periodontal disease in the upper anterior teeth. It will be noted that five of these, designated by an asterisk, are open-bite cases, accompanied by the tongue-thrusting syndrome. It is suggested that in these open-bite cases the force of the tongue is dissipated through the space afforded rather than against the hard palate and the teeth themselves. The tongue in these cases can be exerting a positive axial force to the teeth in the periodontium and therefore the tongue exerts a functional stimulus much as normal occlusion might. These teeth showed normal gingival tissues, and the roentgenographic examination reveals a continuous lamina dura and supporting bone which exemplifies the finding of teeth in occlusion rather than teeth lacking function.

Fig. 5. Patient 25; male; 34 years. Excessive bone resorption and pocket formation on the lingual surface of anterior teeth.

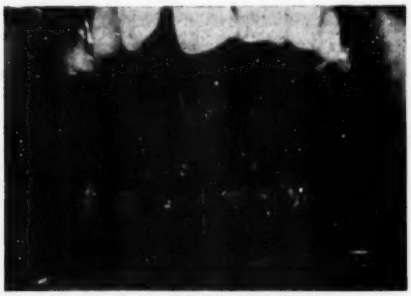

Fig. 4. Tongue at rest. Moderate macroglossia.

DISCUSSION

In the consideration of the relationship of tongue-thrusting to the causative features of periodontal disease, the conditions must be divided into two groups—one, that which is correlated with the syndrome exemplified in our chart of hyposensitive palate, tongue-thrusting and macroglossia and two, that in which tongue habits have been acquired in childhood or adult life as a result of some other stimulus. In both groups, however, we feel that the tongue exerts a very definite force, either repeatedly in the swallowing act as in group 1, or in exercise of the habit as in group 2. The results in both groups however may be identical, the difference may only be in the suggested therapy or correction. The force applied by the tongue is against the long axis of the teeth rather than in the direction of physiologic resistance to force. It might very well be worthy of consideration that this force, exerted laterally, is then further accentuated by a reciprocal occlusal force and aided by the action of

the lips resulting in a jiggling action of the teeth. These teeth then pushed out of position are also subjected to further periodontal trauma in irritations of excursions of food and weak contacts.

In all cases, whether the tongue-thrusting is associated with hyposensitive palate

Case No.	*Age*	*Sex*	*I*	*II*	*III*	*IV*
# 1	42	F	—	X	X	—*
#20	36	F	X	X	X	—
#37	31	F	X	X	X	—
#29	20	F	X	X	X	—*
#30	53	M	X	X	X	—*
#31	52	M	X	X	X	—*

Key to chart: X denotes affected, — denotes not affected, * denotes open-bite cases when anterior teeth do not occlude in any excursion.

Fig. 6. Chart two lists cases in which no periodontal involvement of the anterior teeth is present. It is to be noted that four of this group are open-bite cases.

or the result of habit, macroglossia seems to develop. Therefore, macroglossia itself can be a contributing cause of periodontal disease or a reason for an increase in its severity. While the teeth are in rest position the tongue can exert a continued force on the lingual aspect of the teeth displacing them away from proper interdigitation. This too can result in jiggling. Equilibration of these cases, in order to correct such a feature, would only be of a *temporary nature*.

In the treatment of cases of tongue-thrusting a thorough analysis and knowledge of the factors present must be made in order to compensate for or eliminate this particular feature. When the cause is definitely determined to be the result of a habit acquired during infancy or childhood the correction must be based on a complete understanding of the condition by the dentist and the patient and desire and cooperation on the part of the patient.

Auto-suggestion can be used in some cases, or definite control of the habit by conscious determination in others. For example: in patient #16 a unique combination of etiological factors was presented.

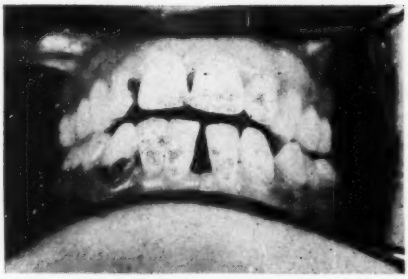

Fig. 7. Patient No. 19; Female age 28 years. Post-operative photograph. Upper right central incisor was displaced labially by tongue thrust. Treated by lingual gingivectomy and Hawley-Hayrake appliance.

The uvula and a portion of the soft palate had been excised during a tonsillectomy. The patient had developed a habit of tongue-thrusting in order to avoid a "gulping noise" upon swallowing. There were four to five millimeter pockets on the lingual surfaces of the upper bicuspids and incisors. A diastema had developed between the upper right cuspid and lateral incisor as the lateral was being forced to the labial with no apparent occlusal trauma in the area. The farther the lateral incisor was forced labially the more intriguing the tongue-thrusting became to the patient. The cause and effect were demonstrated to the patient and after the discussion the patient corrected the habit by auto-suggestion. A later observation showed a great improvement.

A more positive method for the correction of improper swallowing has been suggested by Whitman[8] and used successfully with orthodontic patients. In order to obtain optimum results the step by step procedure should be followed. It is as follows:

GENERAL DIRECTIONS FOR CORRECTION OF IMPROPER SWALLOWING AND TONGUE PERVERSION (Whitman)

1. Have the patient hold a mirror so that he can watch himself.

2. Have him swallow as he normally does, then show him how his tongue comes

between his teeth. Use everyday language that the patient will understand. (Speak to the patient as a friend and not as a censor.)

3. Now take a small, irregular piece of Life Saver, Sen-Sen, or Charms.

4. Take a blunt instrument and tap the posterior part of the hard palate, in the midline, about one-eighth of an inch anterior to its posterior border. In this way, the patient will know where the Life Saver is to be held.

5. Place the small piece of hard candy on the dorsum of the tongue. When the tongue is relaxed and contained within the boundaries of the lower teeth, the piece of candy should be in line with the distal surface of the last erupted molars.

6. The patient then is told to hold the piece of Life Saver on his tongue as you have placed it; then to place it against the spot on the roof of his mouth that you just poked with the blunt instrument.

7. The patient will see that his tongue does not protrude between the teeth if this is done properly.

8. If the swallowing has been done improperly on the first try, the only casualty will be that he will swallow the small piece of Life Saver.

9. Start over again and have the patient sit and practice this for a while before he is dismissed.

10. Go over these directions carefully with the patient, because the patient will need your help and encouragement. You will find that in an open-bite case that is due to a perverted tongue habit the bite will close considerably in a few weeks time if the training is practiced. If the patient has a lisp it will also help overcome that.

11. Speech training, especially the mechanics of sounding S, T, and D, is of great help in tongue habits.

Corrective treatment of tongue-thrusting cases can be made by constructing suitable appliances. These appliances can be designed to re-educate the patient to, in the instance of group 2, correct a habit previously formed and in group 1 to re-educate the patient to a swallowing technique that does not apply forces to the teeth beyond their ability to tolerate.

Various kinds of appliances can be constructed; one a full palate acrylic appliance, which is carried to the lingual cingula of the anterior teeth and closely adapted to the gingival area of the posterior teeth. In the area of the anterior palatal portion a mechanical reminder such as a curtain crossing the palate from cuspid to cuspid or an inverted vault designed as a place to position the tip of the tongue in the swallowing act can be a reminder to correct the habit. In other instances the same basic appliances can be made with a wire "Hayrake" which is used in habit correction by pedodontists. This device, while a bit harsh, will tend to assist the adult in habit correction. The often used Hawley appliance can be used effectively to reposition teeth which have been pushed labially, and at the same time contain a device to correct the swallowing technique or eliminate the untoward habit. Case #19 was treated by the use of a "Hayrake" in cooperation with a Hawley appliance. This case is illustrated by Fig. 7. Sixteen months after initial treatment, in conjunction with periodontal treatment, the patient had controlled the habit, the tooth now remains in its proper position and the periodontal condition is in status quo.

Control treatment can be used in the case of partial denture prosthesis such as with patient #17. In this instance the upper right first bicuspid, cuspid and lateral incisor were missing as were the upper left bicuspids. The patient wore a partial denture that had ill fitting clasps on the upper right central and left cuspid. The posterior clasps were adequate. The periodontal problem was an acute lingual lateral abscess on the right central incisor and a lesser pocket on the cuspid.

The periodontal condition was success-

fully treated. The upper left cuspid and lateral incisor were splinted together with three-quarter crowns and in the lingual embrasure a half-round orthodontic tube was soldered in line with the path of insertion of the new prosthesis which contained a corresponding rod. For the upper right incisor a lingual fossa inlay was placed in which a positive seat was prepared for a rest for the partial. This appliance prevents the labial displacement of the anterior teeth and transmits the masticating forces in the long axis of the teeth.

In the past open-bite cases and malformations as a result of tongue habits have been treated by pedodontists and orthodontists and recognized by them in relation to malocclusion. The periodontist however has not paid sufficient attention to these malfunctions as part of treatment plan to eliminate all etiologic factors present in his cases. The periodontist often sees cases that are not necessarily accompanied by an open-bite and therefore misses glaring tongue habits. Cases in which the anterior teeth appear to be relatively in normal occlusion make the problem difficult to recognize unless his attention is pointed toward it. The periodontist is dealing with a tongue that is much more developed and therefore more difficult to control. In these cases he must realize that the possibility of macroglossia and tongue-thrusting in the adult is accentuated. There are diagnostic features which seem to accompany the finding of tongue-thrusting, whether it is associated with hyposensitive palate or simple habit; they are (1) extensive bone loss on the lingual surface of the maxillary bicuspids, cuspids and incisors and less frequently in the mandibular anteriors, (2) consistent finding of a labial displacement of the incisors and diastema formation without apparent occlusal trauma.

CONCLUSION

A group of 32 patients were observed with periodontal disease in which anomalies of the tongue seemed to be a contributing factor. Upon closer observation two types of tongue thrusting were found to exist—those associated with a hyposensitive palate and those caused by an acquired habit. In the instance of the hyposensitive palate an inevitable syndrome occurs, that is as a result of the hyposensitivity of the uvula the ordinary sequence of events in swallowing is disturbed, and the tongue is forced to compensate for this inadequacy. The tongue-thrusting therefore results with an unusual force and is therefore accompanied by macroglossia. The periodontist must be alert to diagnose the presence of abnormal tongue-thrusting and macroglossia and direct his attention to its correction to the best of his ability, as part of his treatment plan. Certain treatment recommendations have been made. At the present time the corrective exercise suggested by Whitman appears to be the most effective.

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Biologic Basis for Correcting Occlusal Disharmonies †

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IT has been more than fifty years since Karolyi[1] (a dentist in Vienna) described occlusal trauma as a factor in periodontal disease. He observed that people with naturally abraded dentitions and no overbite did not develop periodontal disease and therefore recommended that the incisal and occlusal surfaces be ground in such a manner that all teeth were in contact in protrusive as well as in lateral functional positions. He called this type of occlusion "sled occlusion" or "sliding occlusion." He seems to have been the first to describe grinding of the teeth at night (bruxism) as a damaging factor in periodontal disease, and attempted to remedy it by the use of a bite-block, placed on a bicuspid and a molar on one side of the lower jaw, worn only at night.

Since the time of Karolyi, the problem of occlusal trauma has been discussed extensively and there are not many in the profession today who will deny the importance of this factor in dentistry. There are three questions which come up again and again—First: Is occlusal trauma a primary etiologic factor in periodontal disease? Second: Is it an aggravating or predisposing factor? Third: What is the biologic basis for correction of occlusal disharmonies?

The first question has to be answered in the affirmative. Occlusal trauma is the primary etiologic factor for one form of periodontal disease, namely, periodontal traumatism. If masticatory forces transcend tissue tolerance, they produce damage to the periodontal membrane and thus to the alveolar bone and cementum. The tissue changes that occur due to this excessive force are hemorrhage, thrombosis, necrosis of the periodontal membrane, leading to resorption of bone in the areas of pressure.[2,3,4] Repair of the damaged tissues on the side of compression occurs by resorption of the bone, mainly from adjacent marrow spaces and reorganization of the lost parts of the periodontal membrane by formation of new bone, cementum and principal fibers.

On the tension side, the collagenous fiber bundles are stretched beyond their functional length and are damaged in the area of the intermediate plexus where normally the rearrangement of the fibers of a bundle takes place. This traumatic disarrangement of the fibers is soon repaired by proliferation of fibroblasts, producing new fibers and cementing substance.[5,6,7] New bone develops on the surface of the alveolus, tending to re-establish the normal width of the periodontal membrane.

Neither on the side of pressure, nor on the side of tension, does the tissue damage lead to inflammation, characterized by cellulare infiltration, much less to pocket formation. Though it has been claimed repeatedly that such tissue damage does lead to pocket formation, no experimental evidence has ever been produced to prove it.

Is occlusal trauma a predisposing or aggravating factor in periodontal diseases? This question again has to be answered affirmatively. If an inflammatory process such as a gingivitis or periodontitis involves a tooth or teeth that are also in traumatic occlusion, we can expect a more severe and a less favorable tissue reaction, especially a spreading of the inflammation into damaged areas of the supporting tissues.[7] Under such aggravated conditions, the response to treatment cannot be expected to be favorable. However, we have to understand without any doubt, that gingivitis, periodontitis, periodontosis, atrophy, hyperplasia are not to be confused with traumatic tissue changes. More often, diseases

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like gingivitis, periodontitis, periodontosis and hyperplastic changes will displace a tooth or teeth and thus expose them to secondary occlusal trauma. Then the described tissue changes of traumatism will aggravate and complicate the primary periodontal condition.

An understanding and appreciation of a non-traumatic or better, a harmonious occlusion has to be based upon the general law of closest correlation of structure and function in any organism, organ, part of an organ or tissue. We have to regard structure and function as two aspects of living things. Thus, we see the beautiful functional adaptation of the supporting dental tissues in alveolar bone, cementum, periodontal membrane and dentogingival junction, fibrous as well as epithelial attachment.

It is also a general biological law that almost all tissues and organs have a range of adaptability, without which they could not survive under the necessarily changing functional demands.

The limits of adaptability vary in different individuals according to the constitution and state of health of each. It depends, therefore, also upon the function of different organs—for instance, on the function of the endocrine glands or reticuloendothelial system, and is expressed in differences of capability of regeneration or reaction to injuries. There cannot be a doubt that if there is such an important interrelation between function and structure, a physiologic function is a prerequisite to good tissue health. As previously stated, all tissues have the ability to adaptation within certain limits, so that changes in functional demands can be met satisfactorily in a negative as well as in a positive direction without impairing the functional effectiveness of the organ.

With an understanding of this basic principle, we approach the problem of occlusion not only from the mechanical aspect, but from the point of view of biology. The fact that the teeth are not fixed in the jaw as if set in concrete, but rather are suspended in their sockets, attached to them by ligaments, permits a certain, slight mobility. The teeth are in a delicately balanced functional relation to each other, within the maxilla and mandible as well as in their occlusal and articular relations. The teeth are continually growing and erupting in occlusal direction. This is accompanied by growth of the alveolar bone at the alveolar margin as well as in the alveolar fundus. The active eruption of the teeth compensates harmoniously for the occlusal attrition which occurs continually. The occlusal movement of the teeth is only one phase of this growth process. The other is the continual mesial migration of the teeth which is a compensation for the attrition of the contact points, keeping the teeth in contact in spite of loss of tooth substance in the proximal areas. In addition to these active movements of the teeth, there is a continuous shift of the epithelial attachment along the surfaces of the teeth from the enamel to the cementum. There is also a coordinated shift in the relation of the intra-alveolar and extra-alveolar parts of the teeth.

LOCAL BIOLOGIC FACTORS

We can consider these factors as local biologic factors in the function of the teeth and supporting tissues. The supporting tissues have to adapt themselves continually to the changing relations between tooth and bone. New fibers have to be produced, mainly in the intermediate plexus, to maintain the functional arrangement of the periodontal membrane. Cementum has to be laid down continually on the root surface to maintain the biologic and functional integrity of the tooth and thus to keep the fibrous attachment intact.[8] Like bone, cementum has a limited lifespan. Unlike bone, it is not replaced, but overlaid by newly-formed tissue. Bone has to be formed behind the moving tooth and in addition, alveolar bone has to be reconstructed according to the new functional demands. One must realize that this entire mechanism is rather complex and can easily be disturbed and complicated by outside

interferences, such as periodontal disease, restorative dental procedures and disharmonies in occlusion.

This delicately balanced mechanism is regulated and, therefore, can also be disturbed and complicated by intrinsic factors such as metabolic disturbances, endocrine imbalance, nutritional deficiencies—all conditions in which normal adaptability of the tissues is impaired. One can easily understand the importance of a well-balanced intrinsic environment and extrinsic factors, if one keeps in mind the fact that the supporting tissues of the teeth are not in a static, but in a continually changing dynamic state. The complicated, balanced changes can only take place if disturbing influences are at a minimum.

In addition to this "dental" approach to the functional problem, we have to take all the other parts of the masticatory apparatus into consideration—the muscles of mastication and the temporomandibular joint. The harmonious relation of the masticatory muscles, temporomandibular joint and occlusal surfaces of the teeth is the most important biologic, functional requirement for a normal masticatory organ.

In the past few years investigations into the structure and function of the temporomandibular joint, especially by J. R. Thompson,[9] Harry Sicher,[10,11,12] and others have made us realize that disturbances of the temporomandibular joint, if not produced by some local injury and inflammatory process, and if not of rheumatoid origin, are due to disharmonies between the teeth, muscles and joint. These, in turn, lead to disturbances in muscle coordination, trismus and pain radiating and referred to more distant anatomic parts such as the head, tongue, neck, etc.

A patient does not have to have periodontal disease, loose teeth, or a cracking or sore temporomandibular joint to be eligible for occlusal adjustment. A harmonious relation between teeth, temporomandibular joint and masticatory muscles is the basic objective of a biologic, functional approach

Fig. 1. Mandible in hinge position. In this position, cusp interferences (premature contacts) prevent the teeth from completely occluding. This is the most frequent occlusal disharmony.

to dentistry. If periodontal disease is present, establishment of a harmony is more than a theoretically sound biologic objective. It is an absolute basic clinical or practical requirement. Working on this premise, one may state the following:

1. The supporting tissues of the teeth are structured according to functional requirements; the better the functional relation between the mandibular and maxillary teeth in every position, the less the strain on the supporting tissues. This assures a most favorable tissue response to changes in functional demands.

2. A harmonious relation between the teeth, temporomandibular joint and muscles of mastication is a prerequisite to a physiologic function.

3. Under conditions of health in structure and function, there is great adaptability in our tissues to changing functional demands. This adaptability can be endangered by either extrinsic or intrinsic factors and it is our duty to eliminate these factors and thus to give adaptability its full chance.

4. Periodontal diseases (gingivitis, periodontitis, periodontosis, atrophy, hyperplasia) are disturbances of the structure and function of the supporting tissues of the teeth. Occlusal disharmonies under such conditions constitute a serious aggravating factor.

In addition to these "biologic principles" to be recognized in the problem of occlu-

Fig. 2. Occlusal position of mandible (median). Teeth are in maximum contact. In this case, the mandible has shifted forward and upward from the hinge position (Fig. 1) into occlusal position on inclined planes.

sion, there are the following objectives to keep in mind:

1. Equilibrate occlusion and articulation of the teeth to harmonize with musculature and temporomandibular joint. We do not intend to create a "sled occlusion" according to Karolyi and we do not imitate natural attrition, but rather eliminate interferences and establish harmony.

2. Bring occlusal forces into direction of long axes of teeth bringing lingual cusps of maxillary teeth into central grooves of mandibular teeth and buccal cusps of mandibular teeth into central fossae of maxillary teeth.

3. Distribute forces in functional positions to largest possible number of teeth. Be sure to eliminate premature contacts on non-functioning side. In a natural dentition, a balancing contact is not a functional requirement.

4. Establish static relation between cusps and opposing fossae.

5. Reduce steepness of cusp inclines—establish similar inclines on all teeth.

6. Reduce size of contact surfaces.

7. Retain sharpness of cusps and establish smooth guiding planes.

8. Do not interfere with rest position of the mandible. Do not increase it by grinding and do not reduce it in reconstruction.

In a proper understanding of this problem, we have to be able to express ourselves in a manner understandable to all. The following nomenclature which is simple and clear has been suggested by Harry Sicher:

"Rest Position" of the mandible: A position independent of the teeth, dictated solely by muscle tonus, depending upon the position of the individual and the state of fatigue.

"Centric Occlusion": An ideal position of the mandible in which all components of the masticatory organ, the temporomandibular joint, muscles and teeth are in harmony. (Our endeavor should be directed toward a clear answer to the questions yet unanswered: where is the mandible, especially the condyle situated in centric occlusion and how it might be possible to register and to duplicate this vitally important position.)

"Terminal Hinge Position": The most retruded position of the mandible which the patient can voluntarily achieve with his musculature (Fig. 1). It is the position in which the mandible is placed if the patient is instructed or guided to open in a pure hinge movement exceeding a few millimeters. If the hinge points of the mandible are determined by clutches contacting only by a central bearing screw and if an extraoral tracer is attached to the clutches, one can see that the terminal hinge position of the mandible coincides with the apex of the gothic arch tracing. (See Figs. 3 and 4.)

"Occlusal Positions": All positions in which teeth are in contact (Fig. 2). It may be "median," right and left "lateral," "protrusive" and possibly "retrusive." These positions are related only to the teeth.

In order that we may proceed with occlusal equilibration in a systematic way, there are certain *"Rules of Grinding"* to be followed:

Hinge position: Grind fossae (cusps—only if they interfere in eccentric).

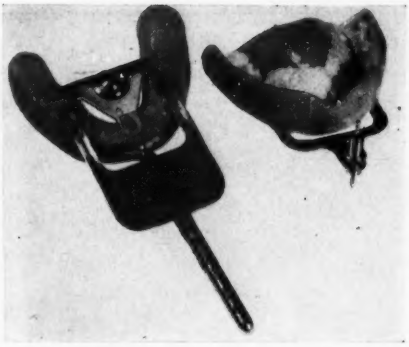

Fig. 3. Plastic clutches with labial bitefork attached for the face-bow. Central bearing screw is attached to lower clutch—platform is on palatal surface of upper clutch. Extra-oral tracers for gothic arch tracing are attached to clutches. Clutches are cemented to teeth with zinc oxide paste which does not set hard.

Grind mesial inclines of upper or distal inclines of lower.

Grind incisal edges of lower incisors.

Protrusive: Grind disto-lingual inclines of buccal upper and mesio-buccal inclines of lingual lower.

Grind lingual inclines of upper front teeth.

Lateral: A. Working side: Grind buccal upper and lingual lower (BU—LL).

B. Non-working side: Grind lingual inclines of buccal lower or buccal inclines of lingual upper but do not take cusps out of functional contact!

General Rule: Never grind a little of the upper and a little of the lower to eliminate an interference.

The technique of occlusal grinding consists of taking occlusally accurate impressions; proper mounting of models in an adjustable articulator; diagnosis; trimming casts and grinding in the mouth. The following steps outline the procedure:

1. Impressions are taken with an alginate or colloid material. Special care must be exercised not to trap air bubbles on occlusal surfaces of the teeth. Models must be made of hard stone.

2. Hinge axis (center of rotation) of the mandible is determined either approximately by palpation: by an average measurement; or by hinge axis determination with an adjustable face-bow. The adjustable face-bow is attached to the lower jaw either by a chin clamp or metal or plastic clutch (Fig. 3) which is cemented to the lower teeth. A clutch is cemented to the upper teeth to serve as a platform for the central bearing screw. Hinge points thus determined (Fig. 4) are marked with an indelible pencil or are tatooed with a 30 gauge needle, using indelible ink.

Fig. 4. Adjustable face-bow (CDF) is attached to cemented clutch. Hinge axis of mandible is determined by opening and closing mandible on the hinge. If hinge axis indicator points do not describe an arc, but rotate in one point, hinge axis is found. Adjustable face-bow's arms can be made shorter or longer; can be raised or lowered.

3. Place wax on bite-fork; have patient bite into wax; attach face-bow to bite-fork and place axis indicators of the face-bow on the tatooed hinge points (Fig. 5). Attach infraorbital pointer to face-bow and point to infraorbital notch. Remove bite-fork with face-bow.

4. Upper model is mounted in articulator on hinge points and infraorbital point on the horizontal plane. Use mounting jig for this operation (Fig. 6).

5. Terminal hinge position of mandible is registered with extra hard wax—two layers with tinfoil between.

6. Narrow wax sticks are softened in water 140 degrees F. Teeth are closed (with mandible in terminal hinge position) with tips of cusps *only* penetrating into the wax and teeth are tapped into wax repeatedly.

7. Lower model is mounted into articulator in terminal hinge position of mandible with the aid of the wax bites.

8. Protrusive bite is registered with wax, similar to that in previous step. Articulator's condylar inclination is set with protrusive record.

Fig. 5. Face-bow is attached to a bitefork that is covered with wax. Hinge point indicators are placed on tatooed hinge points; infraorbital indicator is placed on infraorbital notch. Upper model is mounted in articulator by the use of this bitefork.

9. Lateral relations of the mandible to the maxilla can be similarly obtained with wax bites, and the articulator set for Bennet movement.

10. Premature contacts and interferences are determined by the use of thin typewriter ribbon, especially made for dental use.

11. Plaster teeth are trimmed with knife according to "Rules of Grinding" and a grinding list is made.

12. Grinding in mouth is done with small stones, using wax strips and thin carbon paper.

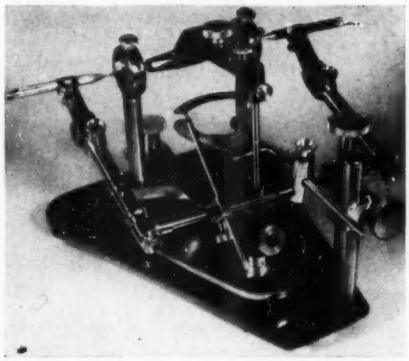

Fig. 6. Face-bow and articulator are placed into mounting jig (CDF). With the aid of the mounting jig, the hinge point indicators of the face-bow can be lined up with the axis of the articulator and the infraorbital pointer lined up on the same plane. Upper model is placed into wax bite on the bitefork and is mounted into articulator with plaster or stone.

The objectives, rules and technique described herein comprise a combination (with individual changes having been made) of those recommended by Clyde Schuyler[13] of New York, Henry Beyron[14] and Gosta Lindblom[15] of Stockholm, Sweden; McCollum[16] and his followers in California; S. W. Brown[17] of Ashtabula, Ohio; Arne Lauritzen[18] of Seattle, Washington; Bertram Downs[19] of Colorado Springs, Colorado, and numerous others.

The biologic principles in question have been intensively studied, especially by Gottlieb and his pupils. Only by proper understanding of the biologic principles, can the mechanical principles be applied to bring about the beneficial results desired.

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For information, write Dean Lester Burket, 4001 Spruce Street, Philadelphia 4, Pa.

Effectiveness of "Berliner Epithelial Scalpel" in Removing the Epithelial Lining of Periodontal Pockets*

by F. W. WERTHEIMER, D.D.S., M.S.D., *Lansing Michigan*

THE question of reattachment has caused much controversy in the dental profession for many years, and is as yet not definitely answered. As far back as 1883, Marshall[1] reported a case of apparent reattachment of the soft tissues to the roots of teeth and regeneration of bone. In 1893, W. S. Younger,[2,3] after explaining his success in closing periodontal pockets, was questioned as to whether there was a contraction of the gum around the teeth rather than a union. Younger said that he meant a union, ". . . and so perfect that the finest instrument could not be inserted between the gum and the tooth without bringing blood." And so began the discussion of whether reattachment is possible, or probable and what the nature of the attachment is.

Orban and Ritchey[4] believe that hope for an organic union between pocket epithelium and the root is faint and the only possibility of success would require the surgical exposure of an intact basal cell layer which according to them is an "unimaginable accomplishment." Orban[5,6] further states that a connective tissue reattachment is possible but not probable.

Although there are those who do not believe that a connective tissue reattachment is possible, many others believe that it can be achieved. However most of the evidence for reattachment has been in the form of clinical measurements in isolated cases with very little histological evidence submitted.[7,8,9,10,11] Ramfjord[12] reported reattachment in experimentally produced pockets on monkeys. His histologic sections showed reattachment even with epithelial cells present in the area of reattachment. Morris[13] has shown evidence of reattachment in surgically created pockets in humans. Shapiro[14] claimed 65 per cent and possibly 85 per cent reattachment to a variable degree in thirty-six pockets. This was a clinical evaluation only. Schaffer and Zander[15] presented histologic evidence of reattachment in several cases and claimed that this study proves that if proper technique is used, a new reattachment of the periodontal tissues to the root surface of the tooth will be accomplished. Glickman,[16] by evaluating the present knowledge pertinent to reattachment, concludes:

". . . if the epithelial attachment and the epithelial lining of the pocket are removed, and the root surface is thoroughly debrided of all degenerated and foreign material, some degree of "reattachment" would occur above the previous area of attachment of the epithelial attachment."

Several methods or techniques, chemical and or mechanical, have been advocated for efficient removal of the pocket epithelium. Antiformin was originally suggested by Hecker[17] in 1913 as a chemical means of ridding the pocket of its epithelial lining. Recently, antiformin has appeared in the literature again.[18,19,20,21,22] Cross stated:[23]

"Pockets which were excised by gingivectomy were treated in this manner, the gingivae subsequently being sectioned and examined; about the same proportion of specimens, whether treated by antiformin or by curettage alone, showed some traces of epithelium present."

Miller and Sorrin[24] reported evidence of the successful use of sodium sulphide to remove the pocket epithelium. Beube,[25] however, showed that the use of sodium sulphide conferred no advantage over careful curettement alone. On the basis of this evidence, it seems that the most efficient means

*Summary of a Thesis Submitted in Partial Fulfillment of the Requirements for the Degree Master of Science in Dentistry at Northwestern University.

for removing the pocket epithelium is by mechanical means.

Very little histological evidence has been presented to give an indication as to how effective curettement is in removing the epithelial lining and attachment in periodontal pockets. Beube[26] reported that a uniform removal of epithelium was achieved by curettement in several specimens that he studied. Waerhaug[27] stated: "Experiments on five human beings showed that also here it had been impossible to remove all the pocket epithelium during ordinary curettage." However Morris[28] recently reported that it is possible to completely remove pocket and attachment epithelium with his technique using McCall Curettes 2R and 2L. This work was confined to labial pockets only.

Since pocket epithelium varies not only in relation to the depth of the pocket in various areas but also in its thickness, the effectiveness of any method cannot be properly evaluated unless serial sections are made. Furthermore, to be of any practical significance, it is felt that both labial or lingual and interproximal pockets must be studied.

Berliner[29, 30] has designed a "self-limiting epithelial scalpel" which he claims will effectively remove the epithelial lining and attachment in periodontal pockets. It is his contention that this instrument will "peel" out the epithelial lining and attachment thereby eliminating the tendency for shredding that occurs with conventional curettes. The technique which he recommends for the use of this instrument is as follows:[31]

"Insert epithelial scalpel *with guards* facing the hard wall—turn at a slight angle and "peel" the soft wall lining off with the blade—excursing back and forth the extent of the pocket area. The tip of the blade is used to remove the epithelial attachment (which if left will preclude a satisfactory result)."

This study was undertaken in an attempt to determine histologically the effectiveness of the "Berliner Epithelial Scalpel" in removing the epithelial lining of periodontal pockets. No attempt was made to show the effectiveness of the instrument in removing the epithelial attachment.

MATERIALS AND METHODS

Periodontal pockets three millimeters in depth or greater were selected where either a gingivectomy or extraction of the teeth adjacent to the pocket was to be performed. In addition, the pocket had to be reasonably accessible. Interproximal pockets were chosen anterior to the first molars and in most cases where a diastema was present. This was found to be necessary in order to adequately and properly insert the "Berliner Epithelial Scalpel" into the pocket and excurse it back and forth in the prescribed manner. If the contact points between the teeth were tight, it was found to be almost impossible to adequately excurse the scalpel completely through the pocket and to remove the interdental papilla intact. Only cases where the clinical signs of inflammation were minimal or nonexistent were selected. It was felt that to properly evaluate the effectiveness of this instrument, both interproximal and labial or lingual pockets should be used.

When the area was anesthetized preparatory to the gingivectomy or extraction procedure, the pocket depth was measured with a calibrated Williams periodontal probe and the bottom of the pocket was marked by a puncture. The "Berliner Epithelial Scalpel" which is manufactured by the Gollobin Dental Company, was then inserted and excursed throughout the pocket in the prescribed manner. The number of excursions made, depended upon the operator's judgment which was governed by the mobility of the tissue and the amount of tissue which appeared to be removed. In all cases an attempt was made to support the tissue as much as possible with the fingers. This was especially necessary around labial and lingual pockets. The lateral excursions were made well beyond the anticipated line of

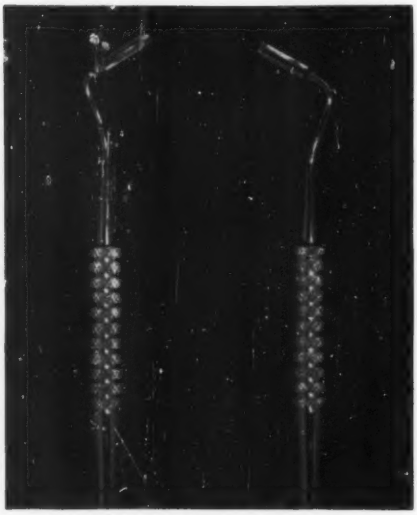

Fig. 1. Berliner Epithelial Scalpel.

incision for the biopsy. Apically the scalpel was forced slightly beyond the bottom of the pocket in cases where a gingivectomy was to be performed. In the extraction cases the scalpel was forced apically until bone was encountered.

In most cases about ten or twelve excursions through the pocket were felt to be enough. In interdental spaces the scalpel was used in one pocket while the pocket on the other side of the papilla was left intact. It was felt that unless this was done as a control, it might be difficult to determine whether the epithelial lining was actually removed by the Berliner scalpel or torn during biopsy removal.

The base of the pocket was then incised to free the tissue. Any gingivectomy knife that would accomplish this in each area was used. In the interdental tissue where orientation would be difficult after fixation and dehydration, a short piece of steel ligature wire was inserted in the buccal or labial surface of the specimen. This was done so that the tissue could be properly oriented for embedding in paraffin. The tissue was immediately placed in Zenker's fluid and fixed for twelve hours. It was then prepared for embedding in paraffin in the usual way. The interdental tissue was serially sectioned mesio-distally and the labial or lingual tissue was sectioned labio-lingually. The importance of serial sections cannot be overemphasized in this type of investigation. One section can present a picture that may not be consistent in other areas of the same tissue. The sections were then stained with hematoxylin and eosin, mounted and examined microscopically for the presence or absence of pocket epithelium.

RESULTS

Thirty biopsies were taken of the soft tissue side of periodontal pockets after the Berliner Epithelial Scalpel had been used. Sixteen of the biopsies were from interdental areas and fourteen from buccolabial and lingual areas. The character or tone of the tissue was graded relatively as firm (+) or flabby (—). The firm tissue was more fibrous and tended to resist displacement whereas the flabby tissue was more edematous, inflamed, and movable. The degree of pocket epithelium removed was evaluated on the basis of complete, good, fair, poor, or no removal. Complete removal is indicated by a lack of any attached epithelial cells in the operated area (See Figure 2). Good removal is indicated by a lack of all but a few deep epithelial projec-

Fig. 2. Intact pocket epithelium on right and complete removal on left. (Case 6 Slide 8)

Fig. 3. Intact pocket epithelium on right and good removal on left. (Case 6 Slide 15)

six slides showing good removal. These represent approximately two hundred eighty serial sections through that pocket.

Of the one hundred ninety-nine slides examined from interdental areas, twenty-eight or 14 per cent showed complete removal. Of the two hundred three buccolabial and lingual slides examined, eighty or 39.4 per cent showed complete removal. It may be concluded that the chance for achieving complete removal of the pocket epithelium is greater in the buccolabial and lingual pockets than in the interdental pockets. In this study no correlation between tissue tone and degree of epithelial removal is apparent.

DISCUSSION

The results of this study show that the Berliner Epithelial Scalpel, in the hands of this operator, will not remove all of the epithelium lining periodontal pockets with any degree of consistency. Although cases were selected with good gingival tone inso-far as was possible, the degree of re-

tions or attached epithelial cells (See Figure 3). Fair removal is indicated by a lack of all epithelium except the projections into the connective tissue and a thin layer of connecting epithelium (See Figure 7). Poor removal is indicated by only a superficial surface removal (See Figure 4). No removal is where the surface epithelium appears to be completely intact (See Figure 8).

The number of slides showing the different degrees of epithelial removal for each case are placed in their respective categories in the tables. Table 1 shows the results from the interdental biopsies while Table 2 shows the results from the buccolabial and lingual biopsies. Each slide contains approximately twenty serial sections. The sections on each slide present about the same picture throughout. Case 1, for example, has two slides showing poor removal, six slides showing fair removal and

Fig. 4. Intact pocket epithelium on right and poor removal on left. (Case 6 Slide17)

TABLE 1

CASE	AREA	TONE	Degree of Epithelial Removal				
			No	Poor	Fair	Good	Complete
1	Interdental	+		2	6	6	
2	"	+			1	6	
3	"	+	7	10	2		
4	"	—		4			
5	"	—			4	12	
*6	"	+		5	5	4	3
7	"	+		3	1	4	1
8	"	+	1		5	7	
9	"	+		2	1	5	6
10	"	—		5		3	2
11	"	—			1	5	7
*12	"	+	1	7	2	1	4
13	"	—				7	
14	"	—	1	3	1	5	3
15	"	—		3	7	7	1
16	"	—		2	4	3	1

*Photomicrographs.

TABLE 2

CASE	AREA	TONE	Degree of Epithelial Removal				
			No	Poor	Fair	Good	Complete
17	Lingual	+					10
18	Labial	—				5	5
19	Labial	—		3	1	2	
20	Lingual	+		1	4	9	4
*21	Labial	+		3	1	6	9
22	Labial	+		3	1		
*23	Lingual	+	13	8			2
24	Lingual	+		2	1	1	5
25	Lingual	+		4	5	10	7
26	Lingual	+		4	1	3	8
27	Lingual	+		2	5	12	8
28	Lingual	+			3	10	2
29	Lingual	+					10
30	Lingual	+					10

*Photomicrographs.

Fig. 5. Intact pocket epithelium on left and poor removal on right. (Case 12 Slide 13)

moval was not noticeably different between the firm and flabby tissues. The cases were also selected for accessibility so as to minimize as many variables as possible. Even with these two variables minimized, the overall epithelial removal was not very complete—14 per cent complete removal in the interdental areas and 39.4 per cent in the buccolabial and lingual areas.

The thickness of the epithelium is one variable that cannot be controlled. This varies from one case to another from several to many cells and very often deep epithelial projections into the connective tissue are observed. The distance between the guards and the cutting blade of this instrument is 0.7-0.9 mm. This was arrived at, when designing the scalpel, by taking the measurement of the average depth of the rete pegs from many sections.[31] However the thickness of the epithelium lining a periodontal pocket varies greatly, not only from pocket to pocket, but also from different areas of the same pocket. Since the Berliner Epithelial Scalpel was designed to cut the average depth instead of the greatest depth of the epithelium, it seems reasonable to assume that much of the thicker epithelial linings would not be completely removed.

It is further noted that the number of slides showing complete removal is greater in the buccolabial and lingual sections than in the interdental sections. Perhaps this difference can be explained by the easier accessibility and greater finger support possible in the labial and lingual areas.

This study also shows that the degree of epithelial removal within a pocket varies from one area to another. This may be due to the different degrees of accessibility within an individual pocket, variations in epithelial thickness within the pocket, and amount of finger support or tissue resist-

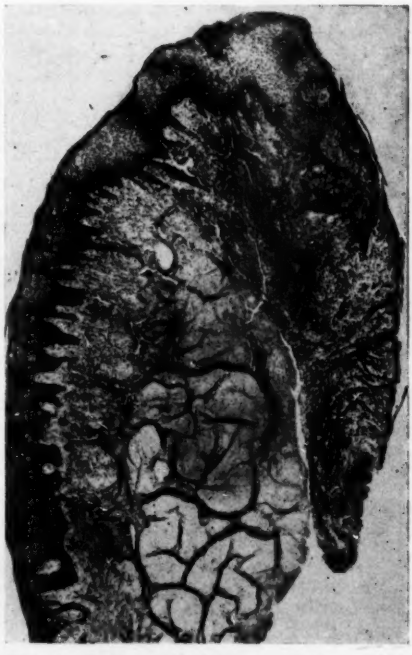

Fig. 6. Pocket epithelium on right showing poor removal. (Case 21 Slide 8)

ance to displacement from one area to another. This variation within a pocket demonstrates the value of serial sections. It would be very easy to select one slide from almost any case to illustrate any one of the different degrees of removal.

If clinical evidence shows that reattachment occurs in most cases after using the Berliner Epithelial Scalpel, then perhaps a complete removal of the pocket epithelium is not necessary in order to achieve reattachment.

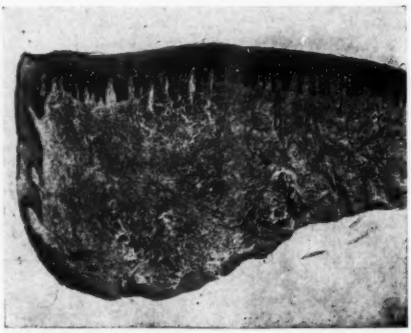

Fig. 8. Pocket epithelium on left showing no removal. (Case 23 Slide 17)

SUMMARY AND CONCLUSIONS

In thirty cases, the Berliner Epithelial Scalpel was used in an attempt to remove the epithelial lining of periodontal pockets. Biopsies were obtained, serially sectioned, and examined microscopically to determine the effectiveness of the scalpel in removing the pocket epithelium. The slides from each case were placed in the categories of complete, good, fair, poor or no removal, depending upon the amount of epithelium remaining in the operated area.

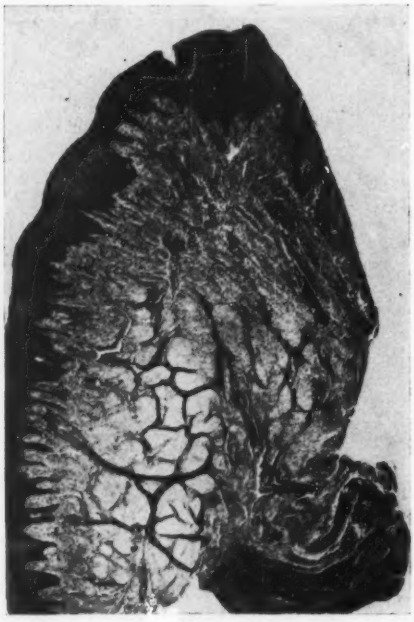

Fig. 7. Pocket epithelium on right showing fair removal. (Case 21 Slide 11)

It was found that pocket epithelium was completely removed in 39.4 per cent of the slides examined in the buccolabial or lingual series and 14 per cent of the slides examined in the interdental series. Only three cases showed complete removal throughout.

If all of the epithelium lining a periodontal pocket must be removed as one of the prerequisites for reattachment, this instrument has little value. It is admitted that many variables do exist in a technique such as this, not the least of which is the operator. If overwhelming clinical evidence shows reattachment after this instrument has been used, then perhaps complete removal of the epithelial lining is not necessary.

"There is nothing so magic about the microscope that makes limited microscopic studies more valuable than overwhelming and good clinical evidence. Arguments, at an academic level, over reattachment should not prevent progress in clinical periodontics."[32]

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A Technic for Reattachment

by FERMÍN A. CARRANZA, D.D.S.,* *Buenos Aires, Argentina*

THE BIOLOGICAL PROBLEM

THE closure of a pathological periodontal pocket actually involves a triple process: (1) a new union between the epithelium and the tooth: Epithelial reattachment. (2) A new insertion of gingival fibers to the tooth: Connective reinsertion. (3) The regeneration and neoformation of bone and periodontal membrane.

The problem of obtaining reinsertion and reattachment is similar to the problem of healing of a wound by first intention.[13] In the periodontium the process is more difficult and complicated than in a wound involving the soft tissues because the dental wall, although it might be vital, has no reparative nor defensive reaction, as the soft tissues have.

Furthermore, the cementum in contact with the periodontal pocket has its connections with the periodontal membrane broken, and is infected. This infection penetrates to varying depths into the organic material of what were perforating fibers. These broken down layers of necrotic cementum, with incrustations of calculus and cracks, bathed in pus which penetrates deeply into the fissures and interstices, and with zones of putrefying and disorganized material[1] are an ideal site for the development and multiplication of microorganisms which are completely isolated from the defensive mechanisms of the body. These layers of infected cementum are an impediment to the union of the tooth to the surrounding vital tissues.

The same as in the problem of healing of a wound by first intention the enemy of reinsertion and reattachment of the periodontal tissues is infection.[3,7]

In our opinion, the lack of vitality of the cementum—which has been exposed to the environment of the pocket for variable lengths of time—is not the cause of failure in reattachment, as contended by Gottlieb.[10] This is proven by cases of transplantation of teeth[2] which were outside the mouth for a year or more—the cementum being non-vital—and in which the epithelium became joined to the cemental surface.

The problem presented by the periodontal pocket is similar to the problem of the root canal in cases of putrescent pulp. The body defensive mechanisms cannot reach the interior of the canal where the infection is located, and the apical granuloma acts as a defensive barrier. As long as the infection of the root canal is not eliminated, the granuloma will persist and the reparation of the periapical tissues will be impossible. The same thing happens in the periodontal pocket: as long as the infection exists, the periodontal granuloma will also exist as a defensive barrier. The lumen and crevices in the cementum have the same significance as the root canal. Only if the necrotic layer is completely eliminated leaving the tooth surface smooth and polished, without crevices, pores or disorganized tissues, will it be possible to sterilize the lumen of the pocket and then to make the union of the gingival tissues to the tooth.

The periodontal granuloma which exists in all pockets[6] delays the healing and the reparative processes of the surrounding tissues. If this granulation tissue, suppurative and old, is not eliminated in the course of the treatment, it must be resorbed by the body. This resorption is sometimes a slow and lengthy process, and will cause the attempts to get reinsertion to fail, as the first stages in the evolution toward reinsertion take place in a few hours with a fibrinous adherence. Furthermore, the

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young granulation tissue formed after the treatment will be in much better condition to develop towards maturity and to promote the reparation of the periodontal tissues.[9] For this reason, the granulation tissue must be eliminated as completely as possible.

The epithelium of the lateral wall of the pocket, infiltrated and more or less degenerated, also conspires against the reinsertion, by not permitting the connective tissue to come into direct contact with the cementum. Only if the epithelial lining of the gingival wall of the pocket is completely eliminated, will the connective tissue be placed in direct contact with the tooth. To attain reinsertion, also complete elimination of the epithelial attachment is necessary.[8] Vestiges of the epithelial attachment will impede the insertion of new fibers to the cementum covered by them. The elimination of the epithelial attachment and of the epithelial lining of the pocket must be complete.

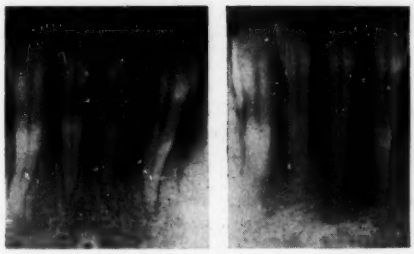

Figure 1
A. Before treatment. Radiographically, vertical bone loss. Clinically, intraalveolar pocket, 6 mm. deep, and a 2 mm. diastema.
B. After treatment. Radiographically, regeneration of the lamina dura and partial repair of the bony crest. Clinically, the pocket was 1 mm. deep and the diastema had disappeared.

THE CLINICAL PROBLEM

The clinical problem consists of finding a way to put the connective tissue of the pocket in contact with the radicular surface within a sterile environment, and to maintain it while the clot is being formed and organized. To this end, the following is necessary:

1st: Complete and total elimination of the epithelial lining of the soft wall of the pocket and of the epithelial attachment. This may be attained by various means: using scalers, curettes, scalpels, etc. The procedures which do the least damage to the tissues are the best as they reduce the possibility of an inflammatory reaction. Caustics[11, 12] are not suitable, as they impose on the tissues the work of removal which conspires against the final result.

2nd: Obtain the complete sterility of the lumen of the pocket and adjacent tissues. The difficulty to attain, and even to maintain this sterility, is the cause of the continuous failures in treatments which attempt reinsertion and reattachment. In order to obtain this sterility we must: (a) eliminate the necrotic cementum, scaling and planing very thoroughly the whole dental surface of the pocket. Not only the subgingival calculus must be scraped off, but the cementum must be scaled until a smooth and polished surface is obtained with no porosities to lodge the micro-organisms. In areas close to the cemento-enamel junction, where the cementum is very thin, sometimes it is necessary to remove the cementum completely, and at times even a layer of dentin. Uncovered dentin, if not infected, will not be a handicap for the deposition of new layers of cementum over it. (b) Removal of the periodontal granulation tissue as completely as possible, curetting the soft wall of the pocket. (c) Eliminate mechanically the necrotic tissues and all septic material filling the pocket until it is completely clean and free of all debris. This clean-up is completed by the more or less abundant hemorrhage which brings away any remaining debris. (d) Sterilize then the lumen of the pocket and adjacent tissues. For this purpose, the use of medicinal agents which do not reduce the vitality of the tissues nor produce necrosis, is recommended.

3rd: Maintain the sterility of the pocket while the clot is being organized. For this purpose, the following is necessary: (a) Splinting of loose teeth: tooth mobility,

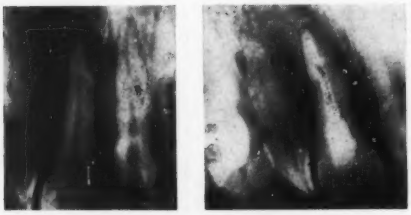

Figure 2
A. Before treatment. Radiographically, bone loss distally and labially to the upper central incisor (left). Periapical lesion. Clinically, 8 mm. pocket in the distal and labial aspects of the tooth.
B. After treatment (18 months). Radiographically, regeneration of the lamina dura and partial repair of the bony crest. Clinically, the labial pocket had disappeared and the distal pocket was 1.5 mm. deep.

which impairs the repose of the tissues, favors the detachment of the epithelium and the penetration of saliva and micro-organisms, with the consequent infection of the clot. In these cases, it is indispensable to fix the teeth by means of ligatures, splints, prosthetics appliances, surgical packs, etc. (b) Avoid the continual masticatory traumatism. A period of absolute rest from mastication is necessary during the first 48 hours; for the first week great care must be taken not to break the first and rather weak union, formed by the fibrin between the tooth and the gingival tissues. (c) Occlusal equilibration: Trauma from occlusion is a mortal enemy of reattachment; each traumatic impact is an attempt against the tranquility of the clot and against the normal reaction of the periodontal tissues.

TECHNIC WITH PENICILLIN[4]

With a very thorough and exact technic, all the necessary requisites for making reinsertion possible can be attained. The weak point in the treatments recommended hitherto, lies in obtaining the sterility of the clot and maintaining it.

For 15 years we have tried a great number of procedures—conservative as well as surgical—and many medicinal agents and substances, all with frankly poor results, until we tried the technic described here. With this technic we are obtaining encouraging results shown by the constant clinical successes and a moderate percentage of X-ray successes.

The powerful antibiotic action of penicillin over a wide bacterial spectrum, together with its innocuity to the living cell makes it ideal for sterilizing the pocket without reducing the vitality nor the normal reactions of the tissues; its pharmacological characteristics allow for immediate action "in situ" as well as prolonged action by systemic administration for the required period of time.

Penicillin locally injected in the neighborhood of the pocket and instilled into it, has a notable clinical action. The usual inflammatory reaction after a vigorous subgingival scaling and curettage beyond the epithelial attachment does not occur when penicillin is used. This places the clot in the best conditions for its organization.

The delaying effect of penicillin over the blood coagulation[14] is not an obstacle in the practice and can be solved by waiting a few moments until the clot is formed.

The only thing we require from the antibiotic is to help the body maintain the sterility of the clot; only use it to this end.

After the equilibration of the occlusal disharmonies, the patient is prepared as for a surgical operation, the area is anesthetized with procaine-adrenalin, and

(1) The root is thoroughly scraped, eliminating the subgingival calculus and carefully planing the cemental surface, reaching the dentin if necessary.

(2) The outer aspect of the pocket is curetted beyond the bottom of the pocket so as to eliminate completely the epithelial attachment, the epithelium of the soft wall of the pocket and the granulation tissue.

These two steps are carried out with iso-

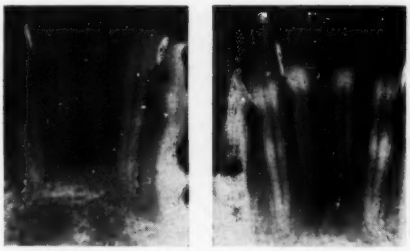

Figure 3
A. Before treatment. Radiographically, the bone has been lost up to the apex. Clinically, distal and buccal pocket, depth 11 mm. Mobility.
B. After treatment (18 months). Radiographically, regeneration of the lamina dura and marked repair of the bony crest. Clinically, pocket depth 1 mm.

lation of the operative area and strict asepsis. Once the scaling and curettage are completed 2 cc. (200.000 units) of potassium penicillin are instilled into the pocket and 3 cc. (300.000 units) are injected in the mucobuccal fold. A slight pressure is exerted until complete hemostasis is obtained, and a clot, as small as possible, is formed. The breach is closed and isolated by means of very soft surgical pack which also, to a certain extent, maintains the isolation of the lumen of the pocket for a variable length of time (2 to 7 days).

For the first three days, 400,000 units of penicillin-procain are given daily by intramuscular route. Only liquids are to be taken for 48 hours and oral hygiene is to be performed daily at the dental office. After a week, the patient starts brushing his teeth with Stillman's technic, avoiding any penetration of bristles. Pockets should not be probed until two months after the treatment.

It must be strongly emphasized that the only thing this technic does is to *place the periodontal structures in the best conditions* for their repair. It is not the action of the antibiotic, which is of short duration but the reparative processes of the body which unite the gingiva to the cementum. This is why it is necessary to eliminate all the factors that can impair the reparative processes and specially to check the occlusion periodically to avoid any traumatic contact that may appear.

The systemic condition of the patient is also of primary importance.[8] Cases with a dystrophic or degenerative disposition (negative bone factor) are less favorable for this type of treatment. Every local or systemic factor suspected to produce or favor this condition should be corrected.

Indications: This technic is specially indicated in cases of intra-alveolar pockets of uniradicular teeth. In this case the bone wall offers a scaffolding that favors the

Figure 4
A. Before treatment. Radiographically, bone loss to the apex. Clinically, 10 mm. pocket, marked mobility.
B. After treatment. Radiographically, new lamina dura and partial repair of the bony crest. Clinically, pocket 1 mm. deep (the gingiva receded 3 mm.).

repair of the bone tissue. However we feel that such a repair is also possible in supra-alveolar pocket. In the latter cases, X-ray visualization is only possible when large regeneration of bone takes place.

CLINICAL AND ROENTGENOGRAPHIC RESULTS

From a total of 103 cases treated with the aforementioned technic over a period of 6 years, we can summarize the clinical results as follows.

(I) Complete failures: 5 cases.

(II) Normalization of the gingival tissues without, or with only a very slight ascent of the top of the pocket towards the crown, and

stabilization of the previously mobile teeth: 57 cases.

(III) Normalization of the gingival tissues with notable reduction of the depth of the pocket by raising of its base crownwards, leading in some cases to complete gingival restitution: 41 cases.

The depth of the pocket was always measured from the base of the pocket to the cemento-enamel junction. For probing the pocket we use blunt and rounded root-canal probes; neither Crane-Kaplan nor Merrit probes nor explorers should be used as their exaggerated thickness leads to very optimistic but false conclusions.

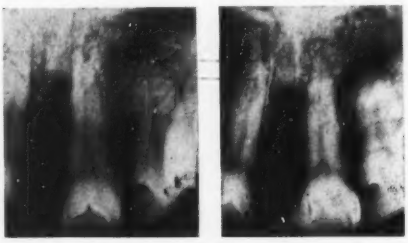

Figure 5
A. Before treatment. Radiographically, marked bone loss. Clinically, upper left central incisor with an 8.5 mm. pocket in the labial and mesial aspects. Upper right central incisor, mesial pocket 7 mm. deep.
B. After treatment (3 years). Radiographically, regeneration of the lamina dura and marked repair of the crest. Clinically, in both teeth, mesial pocket 2 mm. deep; labial pocket depth 0.

The X-ray results were the following:

(1) No variations: 66 cases.

(2) Condensation of the bony outlines: 6 cases.

(3) Formation of a new lamina dura: 3 cases.

(4) Marked repair of the osseous breach: 12 cases.

Sixteen cases did not return after the three months check-up so that X-ray results were not verified.

Whenever new bone formation was proved radiographically, normalization of the gingival tissues and great reduction or disappearance of the pocket by evident crownwards ascension of the base of the pocket, were present. However, there were a great number of cases in which the clinical results were excellent and showed no radiographic improvement.

The earlier signs of X-ray improvement were seen in one case, 7 months after treatment. However, in most cases radiographic changes were seen a year or more after the treatment.

EVALUATION OF THE PROOFS OF REINSERTION

Reattachment proofs can be clinical, radiographic or histological.

Clinically, only the normalization of the gingival tissues, the reduction of the depth of the pocket and the disappearance of its sequelae, can be proved. However, when it is necessary to *prove* the reinsertion, it is a prerequisite to be sure that the disappearance of the pocket is not due to retraction of the gingival tissues but to crownwards ascension of the pocket. A very thorough control of the location of the bottom of the pocket and the distance between the bottom of the pocket to a fixed point of the crown, is necessary for this purpose. And even then, in spite of all

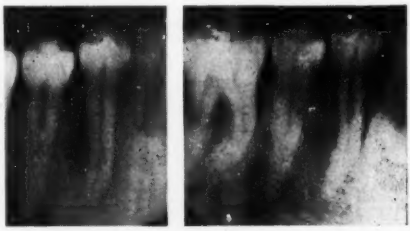

Figure 6
A. Before treatment. Radiographically, vertical bone loss to the apical third. Clinically, labial pocket 11 mm.; distal pocket 10 mm.
B. After treatment (3 years). Radiographically, regeneration of the lamina dura and complete repair of the crest. Remains a moderate widening of the periodontal membrane space.

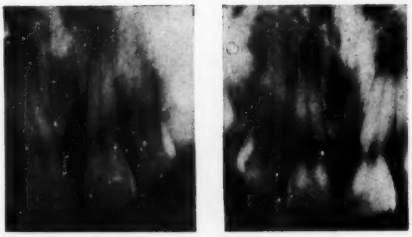

Figure 7
A. Before treatment. Radiographically, vertical bone loss to the middle third of the root. Clinically, labial and mesial pocket, 9 mm.
B. After treatment (3 years). Radiographically, complete repair of the bony crest. Clinically, no pocket.

this care, the clinical proof alone is insecure and as a documentary proof has only a relative value.

Histopathological proofs in experimental animals are very interesting but the real picture of the experimental lesion and the field in which it develops are not similar to human periodontal disease. Artificially produced lesions are not the same as human periodontal disease in that the former lacks all the accumulation of causes that characterize the latter. Histopathological proofs are difficult to carry out in man and should anyway, consist of a thorough comparison of pre-treatment clinical findings with the after-treatment microscopic observations.

In view of the great difficulties in obtaining clinical and histopathological proof, one should lean more towards improving the roentgenographic proofs and evaluating all their possibilities.[5]

It has been said that the radiographic evidence of reattachment is of little value. This is no doubt true when poor X-rays, with different angulations are considered. But if good pre- and post-operative radiographs with the same or very similar angulation and the same intensity are considered, the margin of error or wrong interpretation is reduced to a minimum.

Alveolar bone reparation in areas where pathological pockets existed previously is a proof that reinsertion has been obtained, and this can be shown in the X-rays.

If together with the X-ray result, we also have clinical disappearance of the pocket and normalization of the gingival tissues we must accept that reattachment has taken place.

SUMMARY AND CONCLUSIONS

1. Epithelial reattachment, connective tissue reinsertion and bone regeneration are possible, and may lead in some cases to complete restitution ad integrum of the periodontium.

2. The enemy of reinsertion is infection.

3. The clinical problem in reinsertion lies in placing the connective tissue in contact with the cementum, and keeping it sterile and tranquil while the clot is being organized.

4. Local and systemic penicillin are valuable aids in reattachment technics.

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The Gingival Sulcus: A Clinical Study of Its Depth

by JACOBY T. ROTHNER, D.D.S.,* AND BERNARD B. SATUREN, D.D.S.,** *Philadelphia, Pa.*

INVESTIGATION of dental literature conveys the information that normally the gingival sulcus depth is 0 mm. to 3 mm. Furthermore, publications of the past repeat that the shallower the gingival sulcus the better, and that the 0 mm. sulcus represents the ideal.

Previous reports on sulcus depth were made as the result of studies of histologic sections. The work was done accurately and finely and is fully appreciated. Other statements on clinical depth were made as observations only, without scientific study or investigation.

After making observations on innumerable patients in private and clinic practice, the authors noted that in no instance was there present a 0 mm. sulcus. It was decided, then, that a careful clinical study should be made to determine the exact findings on sulcus depth.

The following standards were established for this investigation:

1. The patient must present a clinically healthy, clean mouth. This standard was set up because we realized that adherent foreign matter of any type might influence sulcus depth. For purposes of determining gingival health, we defined the normal gingiva as being light pink in color, stippled, knife-edged, firm and closely adherent to the teeth, forming an even parabolic curve, with the interdental papillae filling the spaces between the teeth to the contact points.

2. Sulci about teeth on the right side only were measured for uniformity.

3. The sulci about the following teeth were measured:

   a. upper right central incisor

   b. upper right cuspid

   c. upper right first molar

   d. lower right central incisor

   e. lower right second molar.

These teeth were selected as being representative of various types, having unlike functions in the arch, erupting at several age levels, and having differences in root anatomy and alveolar bone support. It was conceded that any or all of these factors

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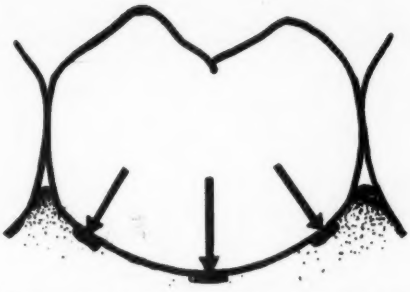

Fig. 1. The periodontal probe is inserted perpendicularly to a tangent of the curve of the gingival margin as shown.

could have a possible bearing on sulcus depth.

4. Sulci measurements were taken on both the facial and lingual aspects of the teeth mentioned. The probe was inserted at three points on each surface (Fig. 1). The proximal surface was not considered.

5. Each measurement was read by two investigators, individually and collectively. In order to maintain uniform standards and to reduce the possibility of error, the same two investigators determined all the measurements.

6. The Williams Periodontal Probe was used as the sole measuring instrument. This instrument was chosen because it was rigid, calibrated, and blunt. The bluntness of the point would not permit it to be forced easily beyond the base of the sulcus giving thereby a false reading for the depth.

Number of measurements		2760
Number of teeth studied		460
Number of patients studied		92
Female	61	
Male	31	
Age Range (years) 12-47.		

Of the great number of patients in the general clinics of the dental school of Temple University, only 92 were found who complied with the standards of the investigation during the period of the survey.

It had been stated heretofore that a sulcus depth of 0 mm. was possible. Our studies, on the other hand, resulted in findings of *measurable* sulcus depths that ranged from 0.25 to 3.0 mm., with none of 0 mm.

In each of the patients studied we found sulcus measurements of varied range. There was, however, one individual where measurements of all sulci examined were the same.

According to our findings sex had no bearing on sulcus depth.

TABLE 1

Tooth	*Surface*	*Range of Depth (mm.)* *Minimum*	*Maximum*	*Average Depth (mm.)*
$\frac{1}{}$	Labial	0.25	2.0	1.104
$\frac{1}{}$	Lingual	0.5	2.0	1.333
$\frac{1}{}$	Labial	0.25	2.0	0.782
$\frac{1}{}$	Lingual	0.25	2.0	0.955
$\frac{3}{}$	Labial	0.5	3.0	1.055
$\frac{3}{}$	Lingual	0.5	3.0	1.491
$\frac{6}{}$	Buccal	0.5	3.0	1.388
$\frac{6}{}$	Lingual	0.5	3.0	1.462
$\frac{7}{}$	Buccal	0.5	3.0	1.309
$\frac{7}{}$	Lingual	0.5	3.0	2.207

TABLE 2

	Tooth	$\underline{1}\vert$	$\overline{1}\vert$	$\underline{3}\vert$	$\underline{6}\vert$	$\overline{7}\vert$
Lingual less than Facial	%	12	14	8	34	4
Lingual equal to Facial	%	42	40	28	26	12
Lingual greater than Facial	%	46	46	64	40	84

Relationship between the lingual and facial sulcus depth

In order to determine a possible relationship between sulcus depth and age, the patients were divided into groups of various age levels. Our findings indicated no differences in sulcus depth between the various groups despite the age range.

By referring to Table 1, the following deductions may be made:

1. The range of sulcus depth of anterior teeth is less than that of the posterior teeth.

2. The average lingual sulcus depth is greater than the average facial sulcus depth for each tooth studied.

3. No deductions could be made from the average measurements of the individual teeth, with the possible exception of the upper and lower central incisors, this being a similarity in the relationship of average labial and lingual depths.

4. The sulcus depth of the facial aspect of the lower teeth measures less than that of the corresponding upper teeth.

5. The upper cuspid is similar to the anterior teeth in the average labial sulcus depth; but similar to the posterior teeth in range of depth.

By referring to Table 2, it may be noted that the percentage relationships between lingual and facial sulcus depths of the incisors are very similar.

Also, one might expect that because the lingual sulcus of the lower molar was deeper than the buccal sulcus, that the reverse should be found about the upper molar because of masticating relationships. However, it was found that this was not exactly true, but by referring to the upper molar in Table 2 one notes the tendency in that direction.

Conclusions: In the examination of sulci of representative teeth of various types in clean and clinically healthy mouths, in no case was a 0 mm. sulcus found; there was some definite and measurable depth in each instance.

Questions for Discussion: In view of this study, did nature intend a gingival attachment without a sulcus? Is it advisable, practical or possible for a 0 mm. sulcus to exist? Why do we not find a gingival attachment without a sulcus? Apparently there is a need for more detailed clinical study of the gingiva about the teeth.

Credit: All the measurements in this study were made by Drs. Stanley Dietz and John Motsko, members of the staff of the Department of Periodontics, School of Dentistry, Temple University.

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The Dimension of Instruments for Removal of Subgingival Calculus*

by JENS WAERHAUG, ARNULF ARNO AND ARNE LOVDAL, *Oslo, Norway*

THE design of our present day instruments for the removal of subgingival calculus can be traced back to those of Riggs.[11] He, more clearly than anybody before him, stressed the destructive effect of calculus in the development of periodontal disease. For the further perfection of the scaling instruments we are indebted to Younger, Good, Harlan, Abbot, Bates, Towner, McCall, and many others. (For review, see McCall.) Great ingenuity has been displayed in devising handy forms allowing all tooth surfaces to be reached, and in this respect a very high standard has been attained. Most of the instruments produced by recognized factories are also well balanced. However, no systematic investigation has been undertaken to decide the most appropriate dimensions of the working end of the instrument. The size of the scaling instruments seems to have been chosen on the basis of clinical experience alone. To be able to give the working end of the instrument the right form and size it is necessary for the designer to be familiar with the normal and pathological anatomy of the gingival pocket, as well as with the ordinary and the extreme dimensions that are met with.

The most common concept of the gingival pocket is based upon the theory about the "epithelial attachment."[6] According to this theory the epithelium is, under favorable conditions, supposed to be firmly attached to the tooth surface all the way to the gingival margin. Under less favorable circumstances a split is believed to form between the epithelium and the tooth surface or within the epithelium, thus forming a pocket. This concept is not in agreement with recent findings,[12,13] which have shown that the bottom of the clinical pocket is to be found at the deepest point of the "epithelial attachment." It has further been shown,[12] that the deepening of the original pocket below the cemento-enamel junction in the majority of cases is related to the downgrowth of calculus, and that the distance between the calculus and the bottom of the deepened pocket is comparatively constant—varying mostly between 0.5 mm and 2 mm. Fig. 1 represents the exact average of 1.2 mm. This means that below the deepest point of the calculus there will always be a pocket of some depth into which the scaling instrument can be inserted, and which accordingly allows the edge of the instrument to reach beneath the most apical part of the calculus. Finally attention was called to the fact, that the distance from the bottom of the pocket to the calculus could be as small as 0.2 mm in deep pockets, where the calculus had grown more than 5 mm below the gingival margin. This tendency is illustrated in Fig. 2 to the left, where the distance is 0.25 mm. The above mentioned

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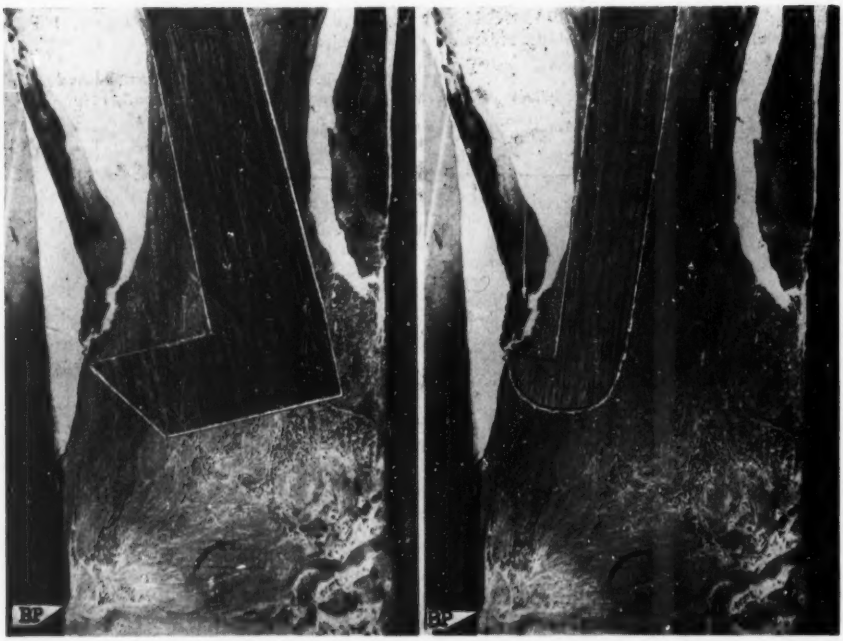

Fig. 1. Right and left photomicrograph of pocket. Enamel disappeared during decalcification. Calculus reaching to 1.2 mm from bottom of pocket (BP), which is slightly deepened below cemento-enamel junction. Artificial split between calculus and soft tissue. Heavy infiltration in connective tissue corresponding to calculus. On left protomicrograph projected photograph of a common small hoe available in the market, both photographed with the same magnification. Scaler measures 1.2 mm from incisal edge to back. In right picture hoe reduced to 0.6 mm.

observations form the basis of the following considerations.

DIMENSION OF INSTRUMENTS

In order to illustrate the size of the instruments in relation to the pockets in which they are going to act, photographs were taken of the instruments with exactly the same magnification as the photomicrographs earlier described. The photographs of the instruments were then mounted on top of the photomicrographs. In Fig. 2 only the outline of the instruments was drawn in.

In Fig. 1 to the left is shown one of the smallest hoes which the authors could obtain in the market. The distance from the cutting edge to the back of the instrument is 1.25 mm. The illustration to the right shows an instrument that has been reduced to less than the half.

In Fig. 2 to the left is drawn the outline of the hoe in its original dimension and in its reduced form. It may be observed that the back of the regular instrument reaches into the alveolar bone. The reduced instrument measures 0.4 mm from edge to back. In the higher magnification to the right there is drawn in an instrument measuring 0.6 mm from edge to back.

The interdental space is unusually narrow (0.5 mm) in Fig. 3. In the left illustration the back of the instrument is projected far into the tooth. The instrument to the right is reduced to 0.4 mm.

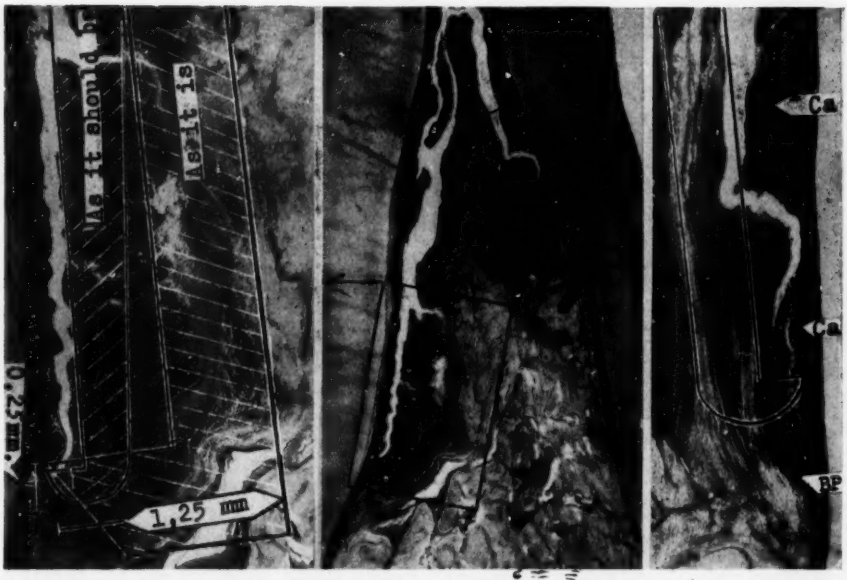

Fig. 2. Left pocket more than 5 mm deep. An 0.1 mm thick layer of calculus reaches to 0.25 mm from bottom of pocket, represented by upper periodontal fibers. Proliferation of epithelium and marked infiltration of round cells in connective tissue corresponding to calculus. Outline of 1.25 mm and 0.4 mm hoes drawn in the photomicrograph. The back of the 1.25 mm hoe reaches the alveolar bone. Pocket on right side 3 mm deep. Distance from calculus to bottom of pocket 0.5 mm. Outline of 0.5 mm hoe drawn in the photomicrograph.

THE INSERTION OF REGULAR AND REDUCED INSTRUMENTS INTO POCKETS

In order to evaluate the influence of a scaler's dimension according to the ease with which it allows itself to be inserted into a deep pocket, the following experiment was undertaken. Into 20 pockets of 5 mm or more, hoes measuring 1.25 mm from edge to back, and hoes measuring 0.4 mm were inserted in the interdental space. The same force (about 400 gm) was applied with both types of instrument. The hoes were pushed down until they met resistance, and in this position they were radiographed. Figs. 4, 5, and 6 show the result. The reduced instrument has penetrated about 2 mm farther down into the pocket than the regular one. These three illustrations are representative of the investigation.

The radiographs show that the edge of the 0.4 mm instrument is projected at the alveolar crest in all three cases. This is, however, no proof that it has reached below the calculus, as faulty projection must be considered. A more thorough control was therefore needed. This control was performed on 30 condemned teeth with pockets measuring 3-10 mm. The hoe reduced to 0.4 mm was inserted into the pocket on one side, while on the other side was used either the 1.25 mm hoe or a curette measuring 1.0 x 0.5 mm. When the instruments met resistance a certain amount of scaling was done on the teeth, so that marks might be left behind on their surface. The teeth were then extracted and stained, and examined under the stereomicroscope.

Fig. 7 shows a tooth with a 10 mm deep pocket scaled with the 0.4 mm hoe. PM is the periodontal membrane, the upper border of which is the bottom of the pocket. The marks made by the instrument are easily seen, some of them are rather deep.

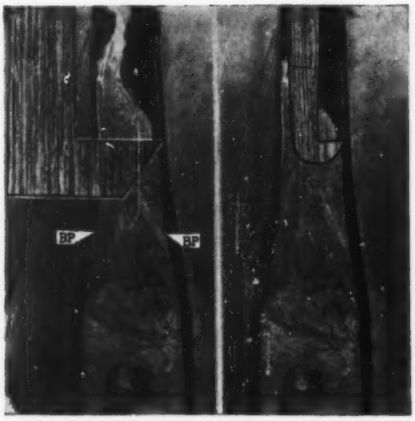

Fig. 3. Interdental space measuring 0.5 mm. Photograph of 1.25 mm and 0.4 mm thick hoes photographed with the same magnification as photomicrographs shown in left and right picture respectively.

On the lingual root (to the left) all the calculus is removed in the deeper part. Higher up and in the bifurcation area rather large remnants are left, Ca. On the buccal root (to the right) the instrument has reached and removed most of the deepest layer of the calculus even if a rather large fragment is left at Ca. X represents a thickened part of the secondary cuticle which is nearly always found just below the calculus. The distance from the calculus to the bottom of the pocket was in this case 0.2 mm.

The consistent result of this experiment was, that the 0.4 mm hoe in all cases had reached below the calculus. The 1.25 mm hoe gave the poorest result, as it only exceptionally reached below the calculus. The curettes gave good results in pockets less than ca. 4 mm, but proved to be less effective in deeper pockets.

DISCUSSION

The comparison undertaken between the photomicrographs and the photographs of the instruments illustrates clearly, that a hoe measuring 1.25 mm from the incisal edge to the back, is in all cases too big to

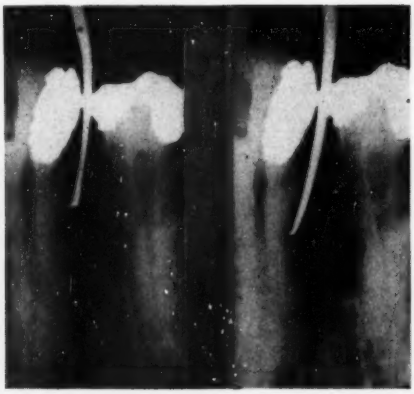

Figure 4

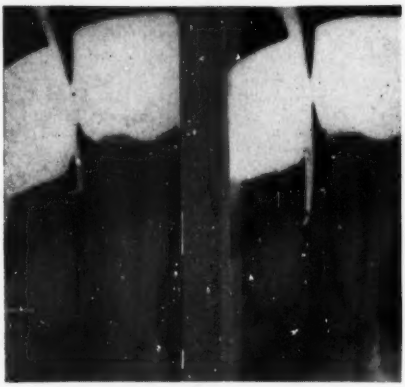

Figure 5

Figure 6

Figs. 4, 5, and 6. Interdental spaces with hoes inserted into the pockets and roentgenographed. To the left is used a 1.25 mm hoe, and to the right, one of 0.4 mm. The reduced hoe reaches about 2 mm deeper into the pocket.

Fig. 7. Tooth which had been scaled with 0.4 mm hoe before extraction. Gingival margin situated at GM. Bottom of pocket situated at upper line of periodontal membrane, PM. Depth of pocket varying between 5 and 10 mm. X indicates deepest line of calculus situated about 0.2 mm from bottom of pocket. Most of the calculus in deepest part of pocket removed, but in other places rather large quantities "overlooked."

be able to get below the deeper strata of the calculus. Even in a case with as favorable anatomical conditions as those shown in Fig. 1, it will be very difficult to press the regular instrument below the calculus, as the back of it will meet too much resistance from the connective tissue of the interdental papilla. With an instrument reduced to about half the size, however, it will be technically possible to reach below the calculus. The part of the pocket situated below the calculus will give ample space for the head of such a hoe. The operator will easily feel the lower border of the calculus, and by pressing the instrument a little further he will recognize a certain space between the deepest point of the calculus and the bottom of the clinical pocket. The fact that there is a pocket of some depth below the calculus actually renders a complete removal of this possible. If the bottom of the pocket had been situated at the deepest point of the calculus, as maintained by Ritchey and Orban,[10] not even the smallest instrument could have reached below this, as is easily seen from these illustrations.

The anatomical conditions are definitely more complicated in Fig. 2 to the left. The regular instrument could not possibly here reach the deepest point of the calculus, as it would even be stopped by the alveolar bone. In such deep pockets very thin instruments have to be used, as the calculus can be expected to lie as near as 0.1 mm from the bottom. The reduced instrument here measures 0.4 mm. Its head should in the present case find place in the 0.25 mm part of the pocket between the calculus and the upper principal fibers. If this space had been 0.1 mm, even a further reduction of the instrument would have been desirable. If a hoe of this size is pressed into the pocket until it meets definite resistance, its incisal edge will most likely get below the calculus, as the soft tissue will to a certain degree give way. In a pocket of the type shown in Fig. 2 to the right it should be technically possible for a hoe measuring 0.5 mm from the incisal edge to the back to effect a good scaling.

Fig. 3 needs no further comments. As it is very common to find interdental spaces of 0.5 mm or even less, special instruments should be constructed for such cases. If hoes are used, they should be reduced to 0.3-0.4 mm. Very often thin sickles give the best results in the interdental space. The present authors use a probe of high quality steel which is ground three-sided. This instrument is very handy, as it is at the same time booth a probe and a scaler. In Figs. 1-3 it will be seen that a rounded back of the instrument will cause a minimum of resistance and damage to the soft tissue.

The experiments illustrated in Figs. 4, 5, and 6 as well as Fig. 7 clearly show the need of small instruments, if these are intended to reach the bottom of the pocket. In the hands of the present authors the 0.4 mm hoe definitely gave the best result. The thin curettes proved to be effective in pockets measuring less than 4.0 mm. But in

deeper pockets their complicated form caused them to meet too much resistance during their insertion. The 1.25 mm hoe must definitely be condemned as useless for removal of subgingival calculus.

Hoes should have an original dimension of 0.6 mm, in which size they are appropriate for rather shallow pockets. According as they wear down on account of sharpening, they will be more and more suited for deep pockets. Only steel of top quality should be used. They should be rounded in both ends of the edge to prevent their making too deep furrows in the teeth. Such furrows cannot entirely be avoided by any of the instruments available, but fortunately they do not represent a decisive obstacle to complete healing.[12]

Fig. 7 illustrates the difficulties met with in very deep pockets. Certainly the 0.4 mm hoe is small enough to be inserted below the calculus, but that does not necessarily mean that the operator will be able to remove all of it. The removal of subgingival calculus is an operation which has to be based entirely on the sense of touch, since a more thorough control can be performed only after the extraction of the tooth. It is, therefore, easy to understand and easy to excuse that even a perfect operator may fail, in spite of the most zealous attempt to succeed. If, in addition to the aforementioned difficulties, he has to work with instruments the size of which does not allow them to reach the deepest part of the calculus, the success is doomed in advance. A control of the scaling technique on teeth that have to be extracted for other reasons is recommended to every practitioner who wants to improve his skill. In the instruction of periodontia such controls should be obligatory.

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Lateral Periodontal Cyst: report of a case.

by W. G. Cross, M.B., M.S., B.D.S., *London, England*

THE classification of cysts proposed by Robinson (1945) includes, under the heading 'development cysts' three forms arising from odontogenetic tissue—the periodontal cyst, the dentigerous cyst, and the primordial cyst.

Robinson classifies periodontal cysts into three types—radicular (apical), lateral and residual. It is with the second of these—the lateral type—that this communication deals.

During the last 25 years a very large number of papers has been published dealing with dental cysts; of these, a few relate to periodontal cysts, but, as far as can be seen, only one case of the type to be described has been reported. This was by Mezrow (1950) who gave a case history, illustrated by diagrams and X-rays, of a lateral periodontal cyst. The condition is not mentioned in any of the leading textbooks of periodontology.

The case to be described was diagnosed radiographically. The patient, a male aged 52, was referred from his general practitioner on account of chronic periodontitis of some upper anterior teeth. Standard full mouth X-rays were taken, and in the ⌈34 region a circular area of resorption was found to be present. This is shown in Figs. 1, 2. The X-ray shows an area of resorption, sharply demarcated, about ¼" in diameter, situated midway down the root, and overlapping both ⌈3 and ⌈4. There are 3-4 mm. of intact crestal bone overlying the area.

Clinical examination revealed little unusual at first glance, except for some toothbrush abrasion cervically ⌈45 (Fig. 3). Closer examination, however, revealed a sinus about 8 mm. deep, with orifice about 1 mm. wide, leading from the gingival crevice mesio-buccally to ⌈4. Firm pressure over the gum between these two teeth caused the extrusion of a cheesy exudate. This was examined bacteriologically and the report was as follows:

Figure 1

Direct film: Gram negative and positive cocci and bacilli with many squames. No evidence of pus.

Figure 2

Figure 3

Culture: Mixed growth of oral flora, including staphylococcus aureus and streptococcus viridans.

Operation: Under submucous infiltration with 2% Xylocaine, a buccal flap was raised and reflected downwards. This exposed the cyst, which was not covered with bone on the buccal aspect (Fig. 4). The cyst was then removed by blunt dissection, and the cavity was found to extend mesially and distally somewhat, in the manner of a figure of eight.

On removal the appearance was as seen in Fig. 5.

PATHOLOGICAL REPORT

"*Macroscopical:* Several small fragments of soft tissue, one with some bone adherent.

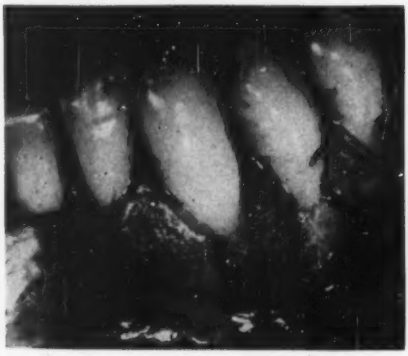

Figure 4

Figure 5

Microscopical: The soft tissue fragments consist of fibrous tissue, in part covered with well formed stratified squamous epithelium and with small detached epithelial masses in the substance of the fibrous tissue.

There is also present a coil of detached keratinized epithelium 4-5 cells in thickness which may be a cyst lining. Probably a periodontal cyst." (Figs. 6, 7.)

Subsequent healing was uneventful. (X-ray three months after operation, Fig 8.)

DISCUSSION

Lateral periodontal cysts appear to be uncommon. In this case, there was a narrow communication between the cyst and the gingival crevice, and although the cyst contents were caseous and not purulent,

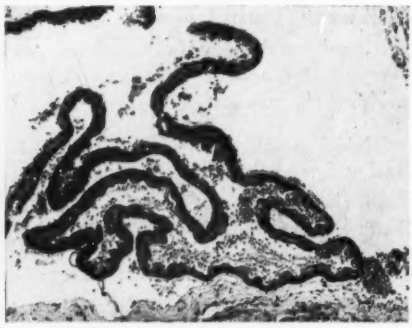

Figure 6

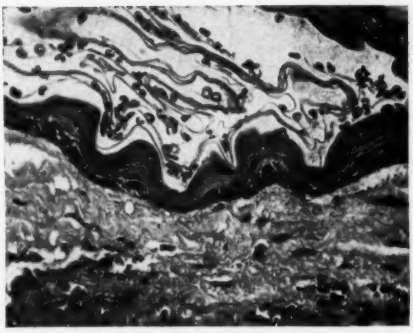

Figure 7

Figure 8

consisting principally of squames, it could clearly have become infected without difficulty and would then have presented as a periodontal abscess. This, it would seem, is a possible mechanism for periodontal abscess formation, and when treatment of a periodontal abscess by curettage is being carried out, the possibility of its being an infected cyst would be overlooked unless the curetted material was examined histologically.

It will be of interest to the author to learn whether periodontal cysts are, in fact, more commonly encountered than a survey of the literature suggests.

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Interpretation of symbols:
* — practice limited to periodontia
† — practice partially limited to periodontia
x — general practice
– — not practicing
d — deceased
( )—date of graduation from dental school

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xDoran, Edward J., D.D.S., B.Sc. (1917)	519 Brisbane Bldg., Buffalo, N. Y.	"	1951
xDrake, M. Llewellynn, D.D.S., M.S., (1915)	1320 Keith Bldg., Cleveland, Ohio	"	1943
xDrellick, Samuel B., D.D.S. (1924)	2929—23rd Ave., Astoria, L. I., N. Y.	"	1948
*Dummett, Clifton Orrin, B.S., (1941) D.D.S., M.S.D., M.P.H.	Box 516, Veterans Adm. Hosp. Tuskegee, Ala.	"	1949
dEdwards, LeRoy Sherman, D.D.S. (1903)	Formerly of Brooklyn, N. Y.	"	1941
xElam, Roy Oscar, D.D.S. (1919)	2102 West End Ave., Nashville 5, Tenn.	"	1942
*Ellis, Clenet R., D.D.S. (1917)	700 First Nat'l. Bk. Bldg., Lexington, Ky.	"	1941
xEmmer, John Wiltz, D.D.S. (1925)	Olivier Bldg., New Iberia, La.	"	1942
xEstry, Julius Edwin, D.D.S. (1927)	333Linwood Ave., Buffalo, N. Y.	"	1942
†Everett, Frank G., M.D., (1941) B.S., D.M.D., M.S.	921 Selling Bldg., Portland 5, Ore.	"	1953
*Fabrizio, Francis J., D.D.S. (1935)	1834 K St., N.W., Washington, D. C.	"	1952
*Fitch, Henry B., Lt. Col., B.S., (1933) D.D.S., M.Sc.D.	Box 6356, Fitzsimons Army Hosp., Denver 8, Colo.	"	1954
*Fitzsimmons, Leslie J., D.D.S. (1923)	144 So. Harrison St., East Orange, N. J.	"	1947
†Foss, Calvin L., D.D.S. (1938)	239 Laurel St., San Diego, Calif.	"	1950
†Fox, Lewis, D.D.S. (1927)	72 West Ave., South Norwalk, Conn.	"	1941
*Friedman, Bernard David, D.D.S. (1912)	25 E. Washington St., Chicago, Ill.	"	1944

*Friedman, Nathan, D.D.S., B.S.D. (1936)	405 N. Bedford Dr., Beverly Hills, Calif.	"	1951
xFriedman, Sidney S., D.D.S. (1922)	1003 Madison Ave., Memphis, Tenn.	"	1942
*Gallagher, J. Wilfred, D.M.D. (1934)	Univ. of N. C. Dental School, Chapel Hill, N. C.	"	1951
xGambill, Edward Little, D.D.S. (1914)	1200 First Nat'l. Bk. Bldg. Lexington, Ky.	"	1942
*Gardner, William M., D.D.S. (1921)	940 Hanna Bldg., Cleveland 15, Ohio	"	1946
xGibbin, Clifford L., D.D.S. (1918)	1120 Temple Bldg., Rochester 4, N. Y.	"	1951
*Giesecke, Max, D.D.S. (1910)	1206 Republic Bldg., Denver 2, Colo.	"	1941
†Gilmore, Earl, D.D.S. (1925)	516 Sutter St., San Francisco, Calif.	"	1944
*Glickman, Irving, B.S., D.M.D. (1938)	483 Beacon St., Boston, Mass.	"	1947
xGold, Arthur, B.S., D.M.D. (1939)	26 Maple St., Springfield, Mass.	"	1953
xGoldhaber, Paul, D.D.S. (1948)	2690 Webb Ave., Bronx 68, N. Y.	"	1954
*Goldman, Henry M., D.M.D. (1935)	482 Beacon St., Boston, Mass.	"	1943
*Golomb, Ida M., B.A., D.D.S. (1945)	Medical Arts Bldg., 57 West 57th St., New York, N. Y.	"	1950
*Gottsegen, Robert, A.B., D.D.S. (1943)	116 East 53rd St., N. Y. 22 and 175 Main St., White Plains, N. Y.	"	1950
–Gracey, Clayton H., D.D.S. (1908)	Glen Lake, Maple City P.O., Michigan	"	1943
*Greenhut, William M., B.S., D.D.S. (1932)	124 E. 84th St., New York 28, N. Y.	"	1946
*Grupe, Harold Earl, D.D.S. (1932)	410 Fisk Professional Bldg., Amarillo, Texas	"	1949
*Hamblen, Tolar N., Jr., D.D.S., M.S.D. (1942)	907 Hermann Prof. Bldg., Houston, Texas	"	1950
*Hart, Virgil Stanley, B.S., D.D.S. (1932)	201 Medical Arts Bldg., Richmond 19, Va.	"	1946
xHebert, Maurice R., D.D.S. (1923)	4255 Indiana Ave., Chicago 15, Ill.	"	1952
*Hicks, Hugh Thomas, D.D.S. (1919)	506 Medical Arts Bldg., Baltimore 1, Md.	"	1942

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*Hileman, Alvin C., B.S., D.M.D. (1943)	1004 Fourth & Pike Bldg., Seattle 1, Wash.	"	1952
*Hirschfeld, Isador, D.D.S. (1902)	730 Fifth Ave., New York 19, N. Y.	"	1941
*Hirschfeld, Leonard S., A.B., D.D.S. (1943)	730 Fifth Ave., New York 19, N. Y.	"	1950
dHoffer, Carl	Formerly Nashville, Tenn.	"	1946
dHoppe, Walt E., D.D.S. (1928)	Formerly Portland, Ore.	"	1949
*Hopper, Craft Ackerman, D.D.S. (1916)	534 Broadway, Paterson, N. J.	"	1941
–Huntington, Roger E., D.D.S. (1911)	Evanston, Ill.	"	1942
*Ingersoll, William Brown, D.D.S. (1932)	1220—16th St., N.W., Washington 6, D. C.	"	1944
†Ingle, John I., D.D.S., M.S.D. (1942)	4719 University Way, Seattle 5, Wash.	"	1951
dJames, Austin F., D.D.S. (1892)	Formerly Chicago, Ill.	"	1941
*James, Edgar, D.D.S. (1919)	1802 David Broderick Tower, Detroit 26, Mich.	"	1951
†Jasper, Elmer A., D.D.S. (1918)	3556 Caroline St., St. Louis 4, Mo.	"	1954
*Johnson, Raymond E., D.D.S. (1918)	824 Lowry Med. Arts Bldg., St. Paul 2, Minn.	"	1941
xJones, Leland D., D.D.S. (1902)	906-907 Bk. of America Bldg., San Diego, Calif.	"	1946
*Jones, Martha Erma, B.Litt., D.D.S. (1919)	525 David Whitney Bldg., Detroit 26, Mich.	"	1943
*Kier, George Paul, D.D.S. (1901)	464 Olive St., San Diego 3, Calif.	"	1943
*Kirkland, George F., D.D.S. (1938)	405 Trust Bldg., Durham, N. C.	"	1948
*Kirkland, Olin, D.D.S. (1902)	722 Shepherd Bldg., Montgomery 4, Ala.	"	1941
*Kramer, Gerald M., D.M.D. (1944)	9 Washington St. at Broad and Nahent Sts., Lynn, Mass.	"	1954
*LaBriola, Robert Rocco, B.S., D.D.S. (1931)	Crenshaw Med. Center, 3761 Stocker St., Los Angeles 8, Calif.	"	1953
*Leabo, Walter, D.D.S. (1909)	706-707 Medical Arts Bldg., Shreveport, La.	"	1941
xLeonard, Harold J., D.D.S., B.A. (1912)	745 Fifth Ave., New York 22, N. Y.	"	1941
*LeRoy, Louis C., D.D.S. (1887)	200 West 59th St., New York, N. Y.	"	1946
xLewis, A. Budner, A.B., D.M.D. (1933)	183 Waterman St., Providence 6, R. I.	"	1950

†Loeb, Virgil, A.B., M.D., D.D.S. (1906)	Beaumont Bldg., 3720 Washington Blvd., St. Louis 8, Mo.	"	1941
dLoftus, J. Leo, D.D.S. (1905)	Formerly of Hartford, Conn.	"	1947
*Lufkin, Arthur W., D.D.S. (1915)	6253 Hollywood Blvd., Hollywood 28, Calif.	"	1948
*Lundquist, G. R., D.D.S., M.S. (1917)	55 E. Washington St., Chicago, Ill.	"	1947
–Lyons, Harry, D.D.S. (1923)	Medical College of Va., Richmond, Va.	"	1941
xLytle, Floyd E., D.D.S. (1930)	125 William Howard Taft Rd., Cincinnati 19, O.	"	1949
dMadison, William James, D.D.S. (1925)	Formerly Washington, D. C.	"	1941
*Mann, Arvin William, D.D.S. (1932)	641 DuPont Bldg., Miami, Fla.	"	1948
dMarre, Leo Francis, D.D.S. (1917)	Formerly of St. Louis, Mo.	"	1942
*McCall, John Oppie, A.B., D.D.S. (1904)	30 West 47th St., New York 23, N. Y.	"	1949
*McKenzie, John Seaborn, D.D.S. (1932)	641 DuPont Bldg., Miami, Fla.	"	1948
*Merritt, Arthur H., D.D.S., M.S., Sc.D. (1895)	580 Fifth Ave., New York 19, N. Y.	"	1941
*Miller, Samuel Charles, D.D.S. (1925)	57 West 57th St., New York 19, N. Y.	"	1941
*Mitchell, Gerald Alex., D.D.S. (1917)	913 Candler Bldg., Atlanta 3, Ga.	"	1943
*Mizell, Daniel Britton, D.D.S. (1919)	213-16 Professional Bldg., Charlotte, N. C.	"	1943
*Morris, Melvin Lewis, B.S., M.A., D.D.S. (1941)	57 West 57th St., New York 19, N. Y.	"	1950
*Moser, Ernest H., D.D.S. (1938)	Lt. Col. D. C. USAF, Far East, c/o 666th Med. Det. (D.C.), APO 20, c/o PM, San Francisco, Calif.	"	1953
*Nabers, Claude L., D.D.S., M.S.D. (1946)	1014 South Texas Bldg., San Antonio, Texas	"	1951
*Nabers, John Moore, D.D.S. (1942)	401 Hamilton Bldg., Wichita Falls, Texas	"	1951
*Nesson, John H., D.M.D. (1922)	120 Boylston St., Boston, Mass.	"	1941
*Ochsenbein, Clifford, D.D.S. (1945)	1212 A Parkway, Austin, Texas	"	1954

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*Ogle, Merle W., D.D.S. (1930)	Lt. Col. U.S.A. (D.C.), Territory of Trieste,	"	1951
xOlder, Lester B., D.D.S. (1934)	2115 New York Ave., Union City, N. J.	"	1954
*Orban, Balint Joseph, M.D., D.D.S. (1938)	30 N. Michigan Ave., Chicago 2, Ill., and 629 N. Nevada Ave., Colorado Springs, Colo.	"	1947
*Parks, Samuel R., D.D.S. (1921)	1505 Medical Arts Bldg., Dallas 1, Texas	"	1946
*Patur, Benjamin, D.D.S. (1932)	36 Woodland St., Hartford 5, Conn.	"	1953
†Paulsen, Albert George, D.D.S., M.S.D. (1945)	1011 N. Highland St., Arlington, Va.	"	1953
*Pendergast, William J., D.M.D. (1944)	45 Bay State Rd., Boston 15, Mass.	"	1953
*Peterson, Lowell N., D.D.S. (1928)	450 Sutter St., San Francisco 8, Colif.	"	1944
†Prichard, John F., D.D.S. (1928)	4121 Camp Bowie Bldg., Fort Worth, Texas	"	1947
*Ray, Harold G., D.D.S., M.S. (1926)	511 Byron St., Palo Alto, Calif.	"	1943
dRich, Celia, D.D.S. (1898)	Formerly Nashville, Tenn.	"	1946
xRitchey, Beryl Thomas, B.S., D.D.S. (1932)	629 N. Nevada Ave., Colorado Springs, Colo.	"	1949
†Robinson, J. Conley, D.D.S. (1931)	903 Hume-Mansur Bldg., Indianapolis, Ind.	"	1943
†Robinson, James Robert, D.D.S. (1939)	Med. Center Bldg., W. Woodruff Ave., Toledo 2, Ohio	"	1952
*Romanov, Irving, D.D.S. (1940)	100 Boylston St., Boston 16, Mass.	"	1953
xRosenthal, S. Leonard, D.D.S. (1922)	1424 Medical Arts Bldg., Philadelphia 2, Pa.	"	1941
*Ross, Ira F., D.D.S. (1942)	25 Halsted St., East Orange, N. J.	"	1953
*Roth, Harry, B.S., D.D.S. (1931)	156 East 52nd St., New York 22, N. Y.	"	1946
*Rothmer, Jacoby Theodore, D.D.S. (1925)	1418 Med. Arts Bldg., Philadelphia 2, Pa.	"	1942
–Rule, Robert William, D.D.S. (1901)	700 N. Orange St., Glendale 3, Calif.	"	1941
*Sanger, C. N., D.D.S. (1934)	3428 W. State St., Milwaukee, Wisc.	"	1953
*Saturen, Bernard Baer, D.D.S. (1932)	1930 Chestnut St., Philadelphia 3, Pa.	"	1942
xSchaffer, Erwin M., D.D.S., M.S.D. (1945)	1153 Med. Arts Bldg., Minneapolis, Minn.	"	1954

*Schluger, Saul, D.D.S. (1931)	30 Journal Sq., Jersey City, N. J. and 116 East 53rd St., New York, N. Y.	"	1943
–Schott, Carlos H., D.D.S. (1903)	1003-1004 Neave Bldg., Cincinnati, Ohio	"	1941
*Scopp, Irwin Walter, B.S., D.D.S. (1934)	4 Stuyvesant Oval New York 9, N. Y.	"	1950
Shack, Aaron H., D.D.S. (1924)	1 Nevins St., Brooklyn, N. Y.	"	1950
*Shapiro, Max, D.D.S. (1939)	Fox Wilshire Theatre Bldg., Wilshire Blvd. & Hamilton Drive, Beverly Hills, Calif.	"	1951
xSilverman, Max, D.D.S. (1923)	5048-49 Jenkins Arcade Bldg., Pittsburgh 22, Pa.	"	1949
xSilvers, Herbert, B.S., D.D.S. (1930)	745 Fifth Ave., New York 22, N. Y.	"	1941
dSinclair, J. A., D.D.S. (1900)	Formerly Asheville, N. C.	"	1941
–Slatten, Richard William, D.D.S. (1947)	Med. Col. of Va., Sch. of Dent., Richmond, Va.	"	1954
dSmith, Rowe, D.D.S. (1923)	Formerly Texarkana, Ark., Tex.	"	1943
*Sorrin, Sidney, D.D.S. (1921)	745 Fifth Ave., New York 22, N. Y.	"	1941
dSpalding, Grace Rogers, D.D.S. (1904)	Formerly Birmingham, Mich.	"	1943
*Sternlicht, Harold C., B.S., D.D.S. (1943)	7218 Staffordshire, Houston, Texas	"	1953
†Stewart, George G., A.B., D.D.S. (1944)	Med. Arts Bldg., 16th & Walnut, Philadelphia, Pa.	"	1952
*Stoker, Ralph E., D.D.S. (1925)	2771 E. Florence, Huntington Park, Calif.	"	1951
xSugarman, Marvin M., D.D.S. (1938)	605 Med. Arts Bldg., Atlanta, Ga.	"	1950
xSwenson, Henry M., B.S., D.D.S. (1942)	658 Fairfield, Indianapolis, Ind.	"	1951
*Swindle, John H., Jr., D.D.S. (1936)	2505 Washington Ave., Waco, Texas	"	1954
†Themann, William A., D.D.S. (1927)	103 Clinton Ave., Newark, N. J. and 80 Green Ave., Madison, N. J.	"	1950
*Thomas, B. O. A., D.D.S., (1935) B.A., M.S., Ph.D.	631 Medical Dental Bldg., Seattle 1, Wash.	"	1951
dTishler, Benjamin, D.M.D. (1904)	Formerly Boston, Mass	"	1941

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*Toye, Alfred E., D.D.S. (1928)	Lt. Col. U.S.A. (D.C.), Tokyo Army Hosp. APO 1052, c/o PM, San Francisco, Calif.	"	1949
xUhrie, Ignatz G., D.D.S. (1919)	903 Security Nat'l. Bk. Bldg., Battle Creek, Mich.	"	1944
–Vaughan, Clarence Bartlett, D.M.D. (1897)	Formerly of Boston, Mass.	"	1947
*Walker, Bernard N., D.D.S. (1932)	301 Hawthorne Lane, Charlotte, N. C.	"	1951
*Walsh, Joseph Richard, D.D.S. (1922)	326 Glen Ave., Short Hills, N. J.	"	1941
xWatkins, Joseph Conrad, D.D.S., ScD., (1900)	1507-1511 Reynolds Bldg., Winston Salem, N. C.	"	1941
–Weeks, Haidee, D.D.S. (1914)	The Wohl, 2111 St. Charles Ave., New Orleans, La.	"	1943
†Weiner, Leonard, A.B., D.M.D. (1940)	2412 E. Elm St., Tucson, Ariz.	"	1954
*Weinmann, Isadore, D.D.S. (1922)	130 Branford Pl., Newark 2, N. J.	"	1947
xWelsh, Arthur, D.D.S. (1918)	818 Starks Bldg., Louisville 2, Ky.	"	1949
xWescott, Randall Livingston, D.D.S. (1923)	636 Church St. Evanston, Ill.	"	1942
*Westbrook, James Craig, B.A., D.D.S. (1932)	1418 Comer Bldg., Birmingham 3, Ala.	"	1943
xWiesstien, Leon Martin, D.D.S. (1919)	922 Rose Bldg., Cleveland 15, Ohio	"	1941
*Williams, Jesse L., D.D.S. (1911)	448 St. James Bldg., Jacksonville, Fla.	"	1943
dWinter, Gordon Richmond, D.D.S. (1919)	Formerly Philadelphia, Pa.	"	1942
xWitkin, George J., A.B., D.D.S. (1942)	45 S. Broadway, Yonkers, N. Y.	"	1954
†Wolfson, Myer D., D.D.S. (1911)	175 Linwood Ave., Buffalo, N. Y.	"	1942
*Wright, Roy B., D.D.S. (1902)	3400 Webster St., Oakland 9, Calif.	"	1941
xYoung, Arthur Price, D.M.D. (1923)	169 Washington St., Wellsley Hills 82, Mich.	"	1941
*Zeretsky, Willa, D.D.S. (1919)	600 Med. Arts Bldg., Grand Rapids, Mich.	"	1941

Announcements

AMERICAN ACADEMY OF PERIODONTOLOGY MEETS IN MIAMI NOVEMBER 4, 5, 6, 1954

The Annual Meeting of the American Academy of Periodontology will be held this year in the Empress Hotel in Miami Beach, Florida, on November 4, 5 and 6. The program, which was published in the July issue of this Journal, includes a symposium on the role of other specialties of dentistry in the prevention and treatment of periodontal disease, a discussion of the concepts of epithelial attachment and wound healing and its relation to various methods of periodontal therapy. Saturday afternoon will be devoted to clinics dealing with periodontal disease. The Annual Banquet of the Academy will be held Friday evening at 7 p.m.

All members of the Academy are urged to attend and anyone interested in the subject may attend as a guest.

THE AMERICAN INSTITUTE OF DENTAL MEDICINE

The next Annual Meeting of the Institute will take place at the Desert Inn, Palm Springs, California, October 31 to November 4, 1954. The faculty will consist of:

William A. Albrecht, Ph.D., University of Missouri, Columbia, Missouri; Charles H. Best, M.D., D.Sc., University of Toronto, Toronto, Canada; Gordon M. Fitzgerald, D.D.S., University of California, San Francisco; Maynard K. Hine, D.D.S., Indiana University, Indianapolis, Indiana; Ernest Jawetz, Ph.D., M.D., University of California, San Francisco; Joseph P. Weinmann, M.D., University of Illinois, Chicago, Illinois.

All Seminar lecturers will participate in a round table forum concerning the application of their subject to the practice of Dental Medicine. Applications and full information may be secured from the Executive Secretary, Miss Marion G. Lewis, 2240 Channing Way, Berkeley 4, California.

AMERICAN ACADEMY OF DENTAL MEDICINE ANNOUNCES MID-ANNUAL MEETING

The American Academy of Dental Medicine will hold its Ninth Mid-Annual Meeting and Luncheon at the Hotel Statler in New York City on Sunday, December 5, 1954, 12:30 p.m. There will be a business meeting for members at 10:30 a.m.

From 2 p.m. to 5 p.m., following the Luncheon, there will be a symposium on BONE. Dr. Harry Sicher will discuss "Bone Formation and Resorption." Dr. J. P. Weinmann will speak on "Variations in the Structure of Bone Tissue and Their Significance in Radiology" and Dr. Lester Cahn will present "The Correlation Between Clinical, Roentgenologic and Histologic Studies in Bone Lesions."

All members and interested dentists and physicians are cordially invited. For reservations and programs address the National Secretary, Dr. William M. Greenhut, 124 East 84th Street, New York 28, N. Y.

OFFICERS OF WESTERN SOCIETY OF PERIODONTOLOGY

Perry A. Ratcliff, D.D.S., President

Arthur Lufkin, D.D.S., Vice-President

Francis J. Conley, B.S., D.D.S., Secretary

Bruce H. Rice, D.D.S., Treasurer

The President of the Western Society of Periodontology is Dr. Perry A. Ratcliff, who is Assistant Professor of Periodontics, Undergraduate and Postgraduate Divisions, School of Dentistry, University of Southern California. He also has the appointment as Demonstrator of Dental Medicine in the College of Dentistry, University of California in the field of research. Dr. Ratcliff was graduated from Indiana University School of Dentistry.

The Vice-President, Dr. Arthur W. Lufkin, is Associate Professor in Oral Histology and Embryology in the College of Dentistry at the University of Southern California. He is well known for his clinics and lectures in periodontology and his many articles. He wrote a book on the "History of Dentistry" in 1938. Dr. Lufkin was graduated from the University of Southern California in 1915.

Secretary Dr. Francis J. Conley, is Director of the Division of Postgraduate Instruction of the School of Dentistry in the University of Southern California. He is interested in both Endodontics and Periodontia, and was graduated from the University of Southern California.

The Treasurer is Dr. Bruce H. Rice, who is on the teaching staff in the Department of Periodontics at the University of Southern California part time and is in practice in Lynwood, California. He was graduated from the University of Southern California in 1954.

The Western Society of Periodontology sponsors an excellent quarterly publication entitled The Journal of the Western Society of Periodontology. The editor of this publication, which features abstracts of current literature dealing with periodontology, is Dr. Max Shapiro from Beverly Hills, California.

The California Academy of Periodontology (Northern California) and the Western Society of Periodontology (Southern California) will hold a combined meeting of the two Societies at Las Vegas, Nevada February 11, 12, and 13, 1955. Dr. Sigurd Ramjford will be the guest lecturer. For further information write to Dr. Ralph E. Stoker, 2771 East Florence Avenue, Huntingon Park, California.

TUFTS COLLEGE DENTAL SCHOOL POSTGRADUATE COURSES

DPG 601—Special Participation Course in Periodontology
8 Mondays on the following dates:
Nov. 22, Nov. 29, 1954; Jan. 3, 10, 17, 24, 31; Feb. 7, 1955
Tuition $200.00 Class limited to 12
Dr. Irving Glickman and Staff

This course emphasized the comprehensive clinical management of the patient with a periodontal problem. Diagnosis determination of prognosis, treatment planning and actual treatment will occupy one-half of the time of the course. The remainder of the time is devoted to lectures and seminars in which the practical aspects of clinical periodontology will be stressed. The course is arranged over a long period of time so that the participants will have the opportunity of following the progress of their patients from the initial visit until the case is completed. The major aim of this course is to acquaint the dentist with the step by step procedure whereby patients with periodontal problems can be managed as part of the practice of general dentistry.

DPG 603—Participation Course in Periodontology
Monday through Friday
April 11-15, 1955
Tuition $150.00 Class limited to 12
Dr. Irving Glickman and Staff

This one-week course consists of lectures and clinical sessions devoted to the various aspects of periodontal disease and their treatment. Half of the time is devoted to clinical practice by the participants under the direction of Dr. Glickman.

DPG 604—Kirkland Gingivectomy Technique
Monday through Friday
May 16-20, 1955
Tuition $125.00 Class limited to 12
Dr. Olin Kirkland

In this course the concept and technique for the Kirkland Gingivectomy operation is presented. Each step in the technique is discussed and demonstrated. The participants operate in the clinic under the direction of Dr. Olin Kirkland.

DPG 605—Occlusal Equilibration
Monday, Tuesday, and Wednesday
February 14, 15, 16, 1955
Tuition $75.00. Class limited to 12
Dr. Edward T. Fischer

This course is devoted to the principles of Occlusal Equilibration as they apply to the treatment of periodontal problems. It will consist of lectures, exercises on models, clinical demonstrations, and participation by students. Diagnosis and case management will be stressed.

Book Reviews

PALAZZI, SILVIO: DIE PARADENTOPATHIEN

This text constitutes a brief review of the periodontium both in normal and pathologic states. After a general discussion of the embryology, histology and physiology of the periodontium, the author emphasizes chiefly inflammatory diseases and their effects on the various component parts of this structure with considerable attention to etiology and pathogenesis. A short chapter on periodontosis is also included as well as a section on treatment of periodontal disease, both mechanical and surgical. The book is only moderately well-illustrated, a lack of clinical photographs being most apparent. This could well form an ancillary portion of the library of those periodontists who read German. (Benno Schwabe & Co., Basel. 1953.) William G. Shafer.

MÜLLER, OSKAR: PULPA-UND WURZELBEHANDLUNG

This textbook, written in German, is a concise but comprehensive treatise dealing with the conservative management of the tooth. The author systematically discusses the treatment, during cavity preparation, pulpitis and its treatment, amputation and extirpation of the pulp, and the root canal filling. The book is well-illustrated with numerous photomicrographs emphasizing the variety of clinical cases which may be encountered and the author ably demonstrates the necessity of a thorough knowledge of the normal histology and histopathology of the tooth by those working in this field of dentistry. An extensive review of the pertinent literature, chiefly German, is appended. (Benno Schwabe & Co., Basel. 1953.) William G. Shafer.

NO APOLOGY NEEDED!

On page 224 of the July issue of the JOURNAL OF PERIODONTOLOGY was published a letter from Dr. Frank G. Everett, in which he suggested that the term "sub-crestal" be considered a preferable term to "intra-bony" or "intra-alveolar." It was called to his attention later that this term had previously been suggested by Dr. George Denton, the scholarly etymologist of the American Dental Association. Dr. Everett then wrote to express his apologies, but Dr. Denton insisted that the term was not original with him and no apology was needed.

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